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(FILE 'HOME' ENTERED AT 08:09:31 ON 16 FEB 2006)

FILE 'AGRICOLA, CABA' ENTERED AT 08:10:23 ON 16 FEB 2006

E ONEAL J/AU  
 L1 1 SEA ABB=ON PLU=ON "ONEAL J"/AU  
 E O NEAL J/AU  
 L2 76 SEA ABB=ON PLU=ON ("O NEAL J"/AU OR "O NEAL J C"/AU OR "O  
 NEAL J D"/AU OR "O NEAL J F"/AU OR "O NEAL J G"/AU OR "O NEAL  
 J K"/AU OR "O NEAL J M"/AU OR "O NEAL J P"/AU OR "O NEAL J  
 R"/AU OR "O NEAL J T"/AU)  
 E WHITE G/AU  
 L3 1205 SEA ABB=ON PLU=ON ("WHITE G"/AU OR "WHITE G A"/AU OR "WHITE  
 G B"/AU OR "WHITE G B B"/AU OR "WHITE G C"/AU OR "WHITE G C  
 II"/AU OR "WHITE G D"/AU OR "WHITE G E"/AU OR "WHITE G F"/AU  
 OR "WHITE G G"/AU OR "WHITE G H"/AU OR "WHITE G J"/AU OR  
 "WHITE G J H"/AU OR "WHITE G K"/AU OR "WHITE G L"/AU OR "WHITE  
 G L JR"/AU OR "WHITE G M"/AU OR "WHITE G N"/AU OR "WHITE G  
 P"/AU OR "WHITE G R"/AU OR "WHITE G S"/AU OR "WHITE G T"/AU OR  
 "WHITE G W"/AU)  
 E WHITE GARY/AU  
 L4 6 SEA ABB=ON PLU=ON "WHITE GARY C"/AU  
 L5 1288 SEA ABB=ON PLU=ON (L1 OR L2 OR L3 OR L4)  
 L6 8558 SEA ABB=ON PLU=ON CRATAEVA OR WILLOW BARK OR SALIX

FILE 'REGISTRY' ENTERED AT 08:13:33 ON 16 FEB 2006

E D-MANNOSE/CN  
 L7 1 SEA ABB=ON PLU=ON D-MANNOSE/CN

FILE 'AGRICOLA, CABA' ENTERED AT 08:13:45 ON 16 FEB 2006

L8 6461 SEA ABB=ON PLU=ON L7 OR MANNOSE  
 L9 2 SEA ABB=ON PLU=ON L8 AND L6  
 L10 48067 SEA ABB=ON PLU=ON PLANT (3A) (EXT## OR EXTRACT?)  
 L11 78 SEA ABB=ON PLU=ON L8 AND L10  
 L12 534012 SEA ABB=ON PLU=ON INFECT?  
 L13 3 SEA ABB=ON PLU=ON L11 AND L12  
 D SCAN  
 L14 61186 SEA ABB=ON PLU=ON POLLEN  
 L15 20 SEA ABB=ON PLU=ON L14 AND L8  
 L16 0 SEA ABB=ON PLU=ON L15 AND L12  
 L17 226 SEA ABB=ON PLU=ON L6 AND L12  
 L18 6083 SEA ABB=ON PLU=ON URIN? (L) L12  
 L19 0 SEA ABB=ON PLU=ON L17 AND L18  
 L20 36 SEA ABB=ON PLU=ON L18 AND L10  
 L21 86878 SEA ABB=ON PLU=ON MEDICINAL (L) (PLANT#)  
 L22 69 SEA ABB=ON PLU=ON L21 AND L18  
 L23 1 SEA ABB=ON PLU=ON L22 AND L8  
 D SCAN  
 L24 5653 SEA ABB=ON PLU=ON URINARY (2A) TRACT?  
 L25 3 SEA ABB=ON PLU=ON L24 AND L6  
 L26 9 SEA ABB=ON PLU=ON L9 OR L13 OR L23 OR L25  
 L27 1 SEA ABB=ON PLU=ON L5 AND L6  
 D SCAN  
 L28 87 SEA ABB=ON PLU=ON L5 AND L12  
 L29 1 SEA ABB=ON PLU=ON L28 AND URIN?  
 D SCAN  
 L30 2 SEA ABB=ON PLU=ON L27 OR L29  
 L31 2 SEA ABB=ON PLU=ON L30 NOT L26

Krishnan Ganapathy 10/691,423

=> fil agricola caba  
FILE 'AGRICOLA' ENTERED AT 08:21:44 ON 16 FEB 2006

FILE 'CABA' ENTERED AT 08:21:44 ON 16 FEB 2006  
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=> d que 126  
L6 8558 SEA CRATAEVA OR WILLOW BARK OR SALIX  
L7 1 SEA FILE=REGISTRY ABB=ON PLU=ON D-MANNOSE/CN  
L8 6461 SEA L7 OR MANNOSE  
L9 2 SEA L8 AND L6  
L10 48067 SEA PLANT (3A) (EXT## OR EXTRACT?)  
L11 78 SEA L8 AND L10  
L12 534012 SEA INFECT?  
L13 3 SEA L11 AND L12  
L18 6083 SEA URIN? (L) L12  
L21 86878 SEA MEDICINAL (L) (PLANT#)  
L22 69 SEA L21 AND L18  
L23 1 SEA L22 AND L8  
L24 5653 SEA URINARY (2A) TRACT?  
L25 3 SEA L24 AND L6  
L26 9 SEA L9 OR L13 OR L23 OR L25

=> d que 131  
L1 1 SEA "ONEAL J"/AU  
L2 76 SEA ("O NEAL J"/AU OR "O NEAL J C"/AU OR "O NEAL J D"/AU OR "O NEAL J F"/AU OR "O NEAL J G"/AU OR "O NEAL J K"/AU OR "O NEAL J M"/AU OR "O NEAL J P"/AU OR "O NEAL J R"/AU OR "O NEAL J T"/AU)  
L3 1205 SEA ("WHITE G"/AU OR "WHITE G A"/AU OR "WHITE G B"/AU OR "WHITE G B B"/AU OR "WHITE G C"/AU OR "WHITE G C II"/AU OR "WHITE G D"/AU OR "WHITE G E"/AU OR "WHITE G F"/AU OR "WHITE G G"/AU OR "WHITE G H"/AU OR "WHITE G J"/AU OR "WHITE G J H"/AU OR "WHITE G K"/AU OR "WHITE G L"/AU OR "WHITE G L JR"/AU OR "WHITE G M"/AU OR "WHITE G N"/AU OR "WHITE G P"/AU OR "WHITE G R"/AU OR "WHITE G S"/AU OR "WHITE G T"/AU OR "WHITE G W"/AU)  
L4 6 SEA "WHITE GARY C"/AU  
L5 1288 SEA (L1 OR L2 OR L3 OR L4)  
L6 8558 SEA CRATAEVA OR WILLOW BARK OR SALIX  
L7 1 SEA FILE=REGISTRY ABB=ON PLU=ON D-MANNOSE/CN  
L8 6461 SEA L7 OR MANNOSE  
L9 2 SEA L8 AND L6  
L10 48067 SEA PLANT (3A) (EXT## OR EXTRACT?)  
L11 78 SEA L8 AND L10  
L12 534012 SEA INFECT?  
L13 3 SEA L11 AND L12  
L18 6083 SEA URIN? (L) L12  
L21 86878 SEA MEDICINAL (L) (PLANT#)  
L22 69 SEA L21 AND L18  
L23 1 SEA L22 AND L8  
L24 5653 SEA URINARY (2A) TRACT?  
L25 3 SEA L24 AND L6  
L26 9 SEA L9 OR L13 OR L23 OR L25  
L27 1 SEA L5 AND L6  
L28 87 SEA L5 AND L12  
L29 1 SEA L28 AND URIN?  
L30 2 SEA L27 OR L29  
L31 2 SEA L30 NOT L26

=> d ibib ab ct 126 1-9;d ibib ab ct 131 1-2

L26 ANSWER 1 OF 9 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved.  
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ACCESSION NUMBER: 2003:40206 AGRICOLA  
DOCUMENT NUMBER: IND23321471  
TITLE: Antiurolithiatic activity of **Salix** taxifolia aqueous extract.  
AUTHOR(S): Vargas S, R.; Perez G, R.M.  
AVAILABILITY: DNAL (RS160.I47)  
SOURCE: Pharmaceutical biology, Dec 2002. Vol. 40, No. 8. p. 561-563  
Publisher: Lisse, the Netherlands : Swets & Zeitlinger, c1998-  
CODEN: PHBIFC; ISSN: 1388-0209  
NOTE: Includes references  
PUB. COUNTRY: Netherlands  
DOCUMENT TYPE: Article  
FILE SEGMENT: Non-U.S. Imprint other than FAO  
LANGUAGE: English

L26 ANSWER 2 OF 9 AGRICOLA Compiled and distributed by the National Agricultural Library of the Department of Agriculture of the United States of America. It contains copyrighted materials. All rights reserved.  
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ACCESSION NUMBER: 1998:42128 AGRICOLA  
DOCUMENT NUMBER: IND21233362  
TITLE: Seedborne fungal contamination: consequences in space-grown wheat.  
AUTHOR(S): Bishop, D.L.; Levine, H.G.; Kropp, B.R.; Anderson, A.J.  
AVAILABILITY: DNAL (464.8 P56)  
SOURCE: Phytopathology, Nov 1997. Vol. 87, No. 11. p. 1125-1133  
Publisher: St. Paul, Minn. : American Phytopathological Society, 1911-  
CODEN: PHYTAJ; ISSN: 0031-949X  
NOTE: Includes references  
PUB. COUNTRY: Minnesota; United States  
DOCUMENT TYPE: Article  
FILE SEGMENT: U.S. Imprints not USDA, Experiment or Extension  
LANGUAGE: English

AB Plants grown in microgravity are subject to many environmental stresses that may promote microbial growth and result in disease symptoms. Wheat (cv. Super Dwarf) recovered from an 8-day mission aboard a NASA (National Aeronautics and Space Administration) space shuttle showed disease symptoms, including girdling of leaf sheaths and chlorosis and necrosis of leaf and root tissues. A *Neotyphodium* species was isolated from the seed and leaf sheaths of symptomatic wheat used in the spaceflight mission. Certain isozymes of a peroxidase unique to extracts from the microgravity-grown **plants** were observed in **extracts** from earth-grown *Neotyphodium*-**infected** plants but were not present in noninfected wheat. The endophytic fungus was eliminated from the wheat seed by prolonged heat treatment at 50 degrees C followed by washes with water at 50 degrees C. Plants from wheat seed **infected** with the *Neotyphodium* endophyte were symptomless when grown under

greenhouse conditions, whereas symptoms appeared after only 4 days of growth in closed containers. Disease spread from an **infected** plant to noninfected plants in closed containers. Dispersion via spores was found on asymptomatic plants at distances of 7 to 18 cm from **infected** plants. The size and shape of the conidia, mycelia, and phialide-bearing structures and the ability to grow rapidly on carbohydrates, especially xylose, resembled the characteristics of *N. chilense*, which is pathogenic on orchard grass, *Dactylis glomerata*. The *Neotyphodium* wheat isolate caused disease symptoms on other cereals (wheat cv. Malcolm, orchard grass, barley, and maize) grown in closed containers.

CT cell cultures; conidia; diagnostic techniques; endophytes; fructose; fungal morphology; glucose; growth; **infectivity**; inoculum density; leaves; **mannose**; mycelium; pathogenicity; peroxidase; plant composition; plant disease control; plant pathogenic fungi; roots; row spacing; seedborne fungi; space flight; spore dispersal; spore germination; sucrose; symptoms; temperature; *triticum aestivum*; xylose; zero gravity

L26 ANSWER 3 OF 9 CABA COPYRIGHT 2006 CABI on STN

ACCESSION NUMBER: 2004:23722 CABA

DOCUMENT NUMBER: 20033209915

TITLE: In vitro and in vivo antiviral properties of sulfated galactomannans against yellow fever virus (BeH111 strain) and dengue 1 virus (Hawaii strain)

AUTHOR: Ono, L.; Wollinger, W.; Rocco, I. M.; Coimbra, T. L. M.; Gorin, P. A. J.; Sierakowski, M. R.

CORPORATE SOURCE: Laboratorio de Biopolimeros, Departamento de Quimica, Setor de Ciencias Exatas, Centro Politecnico, Universidade Federal do Parana, Cx.P. 19081, Jardim das Americas, CEP 81531-990 - Curitiba, PR, Brazil. mariarita.sierakowski@ufpr.br

SOURCE: Antiviral Research, (2003) Vol. 60, No. 3, pp. 201-208. 50 ref.

Publisher: Elsevier Science B.V. Amsterdam

ISSN: 0166-3542

PUB. COUNTRY: Netherlands Antilles

DOCUMENT TYPE: Journal

LANGUAGE: English

ENTRY DATE: Entered STN: 20040206

Last Updated on STN: 20040206

AB Two galactomannans, one extracted from seeds of *Mimosa scabrella*, having a **mannose** to galactose ratio of 1.1, and another with a 1.4 ratio from seeds of *Leucaena leucocephala*, were sulfated. The products from *M. scabrella* (BRS) and *L. leucocephala* (LLS) had a degree of sulfation of 0.62 and 0.50, and an average molecular weight of 620x103 and 574x103 g mol<sup>-1</sup>, respectively. Their activities against yellow fever virus (YFV; BeH111 strain) and dengue 1 virus (DEN-1; Hawaii strain) were evaluated. This was carried out in young mice following intraperitoneal **infection** with YFV. At a dose of 49 mg kg<sup>-1</sup>, BRS and LLS gave protection against death in 87.7 and 96.5% of the mice, respectively. When challenged with 37.5 LD50 of YFV, mice previously inoculated with BRS+virus or LLS+virus, showed 93.3 and 100% resistance, respectively, with neutralization titers similar to mice injected with 25 LD50 of formaldehyde-inactivated YFV. In vitro experiments with YFV and DEN-1 in C6/36 cell culture assays in 24-well microplates showed that concentrations that produced a 100-fold decrease in virus titre of YFV were 586 and 385 mg l<sup>-1</sup> for BRS and LLS, respectively. For DEN-1 they were 347 and 37 mg l<sup>-1</sup>, respectively. Sulfated galactomannans, thus demonstrate in vitro and in vivo activity against flaviviruses.

CT antiviral properties; dengue; disease models; galactomannans; galactose;

in vitro; laboratory animals; **mannose**; **plant extracts**; seeds; sulfate; yellow fever

L26 ANSWER 4 OF 9 CABA COPYRIGHT 2006 CABI on STN

ACCESSION NUMBER: 2003:197303 CABA

DOCUMENT NUMBER: 20033174911

TITLE: Predicting the cold hardiness of willow stems using visible and near-infrared spectra and sugar concentrations

AUTHOR: Lennartsson, M.; Ogren, E.

CORPORATE SOURCE: Umea Plant Science Centre, Department of Forest Genetics and Plant Physiology, Swedish University of Agricultural Sciences, 901 83 Umea, Sweden.

SOURCE: Trees: Structure and Function, (2003) Vol. 17, No. 5, pp. 463-470.

Publisher: Springer-Verlag. Berlin

ISSN: 0931-1890

URL: <http://www.springerlink.com/app/home/contribution.asp?wasp=b5crlc2bpm5qnv4e4m2u&referrer=parent&backto=issue,11,11;journal,3,53;browsepublicationsresults,468,500;>

PUB. COUNTRY: Germany, Federal Republic of

DOCUMENT TYPE: Journal

LANGUAGE: English

ENTRY DATE: Entered STN: 20031209

Last Updated on STN: 20031209

AB Closely related, fast-growing clones of willows from northern/continental and southern/maritime origins were assessed for their levels of cold hardiness. Assessments were made during active growth and, subsequently, during cold hardening at mean temperatures of 3[deg]C (the COLD regime) and 8[deg]C (the MILD regime). The onset of hardening was triggered simultaneously in all clones by administering a drastic day length reduction on the first day. The northern/continental clones showed consistently higher rates of hardening than the southern/maritime ones. This was particularly true under the COLD regime, suggesting that their hardening was less sensitive to low temperatures. The stems' visible and near-infrared absorption spectra, and concentrations of ten major soluble sugars, were also determined. Multivariate analysis revealed that spectral data could predict up to 96% of the variation in cold hardiness, when the analysis was restricted to the MILD regime and the data corrected for irrelevant systematic information. Possible direct links between spectral changes and chemical changes are discussed. Multivariate analysis also revealed that sugar concentrations could be used to predict up to 73% of the variation in cold hardiness. Different sugars displayed different patterns of variation during hardening. Concentrations of **mannose** and myo-inositol both decreased, whereas concentrations of galactose, sucrose, maltose, raffinose and stachyose all increased, but at different times. Dry matter increased markedly during hardening, so expressing the concentrations of sugars relative to dry matter does not provide an accurate measure of the amounts present.

CT chemical composition; cold hardening; cold resistance; galactose; growth; maltose; **mannose**; myo-inositol; raffinose; spectroscopy; stachyose; sucrose; sugar content; temperature

L26 ANSWER 5 OF 9 CABA COPYRIGHT 2006 CABI on STN

ACCESSION NUMBER: 2003:67304 CABA

DOCUMENT NUMBER: 20033029801

TITLE: Antiuro lithiatic activity of **Salix taxifolia** aqueous extract

AUTHOR: Vargas S., R.; Perez G., R. M.

CORPORATE SOURCE: Laboratorio de Investigacion de Fitofarmacologia,  
Universidad Autonoma Metropolitana-Xochimilco,  
Mexico D.F., Mexico. rmpg@prodigy.net.mx

SOURCE: Pharmaceutical Biology, (2002) Vol. 40, No. 8, pp.  
561-563. 10 ref.  
Publisher: Swets & Zeitlinger. Lisse  
ISSN: 1388-0209

PUB. COUNTRY: Netherlands Antilles

DOCUMENT TYPE: Journal

LANGUAGE: English

ENTRY DATE: Entered STN: 20030502  
Last Updated on STN: 20030502

AB The aqueous extract of the bark of *S. taxifolia* collected from Durango,  
Mexico and commonly known as taray was tested for antilithiatic and  
diuretic activities. Urolithiasis was experimentally induced by  
implantation of a zinc disc in the urinary bladder of rats. A significant  
decrease in the weight of the stones was observed after treatment in  
animals with the aqueous extract. This extract caused an increase in the  
24 h urine volume.

CT bark; bladder; chemical composition; diuretics; medicinal plants;  
pharmacology; plant composition; plant extracts; traditional medicines;  
**urinary tract; urinary tract**  
diseases; urine; urolithiasis

L26 ANSWER 6 OF 9 CABA COPYRIGHT 2006 CABI on STN

ACCESSION NUMBER: 2001:653 CABA

DOCUMENT NUMBER: 20000316729

TITLE: Control of urinary risk factors of stones by betulin  
and lupeol in experimental hyperoxaluria

AUTHOR: Vidya, L.; Varalakshmi, P.

CORPORATE SOURCE: Department of Medical Biochemistry, Dr A.L.M. Post  
Graduate Institute of Basic Medical Sciences,  
University of Madras, Taramani, Chennai 600 113,  
India.

SOURCE: Fitoterapia, (2000) Vol. 71, No. 5, pp. 535-543. 34  
ref.  
ISSN: 0367-326X

DOCUMENT TYPE: Journal

LANGUAGE: English

ENTRY DATE: Entered STN: 20010111  
Last Updated on STN: 20010111

AB Urolithiasis, the process of formation of stones in the kidney and the  
**urinary tract**, is the major clinical manifestation of  
hyperoxaluria. Crystal deposition, as indicated by increased stone-forming  
constituents in urine, such as calcium, oxalate and uric acid, and  
decreased concentration of inhibitors, such as magnesium and  
glycosaminoglycans, was observed in pyridoxine-deficient hyperoxaluric  
rats. Renal tubular damage was indicated by increased excretion of enzymes  
such as alkaline phosphatase, lactate dehydrogenase, [gamma]-glutamyl  
transferase, [beta]-glucuronidase and N-acetyl glucosaminidase.  
Fibrinolytic activity was reduced. Administration of pentacyclic  
triterpenes such as lupeol (isolated from *Crataeva nurvala*  
[*Crateva religiosa*] bark) and its structural analogue, betulin, to  
hyperoxaluric rats at 35 mg/kg per day, p.o., for 21 days minimized the  
tubular damage and reduced the markers of crystal deposition in the  
kidneys; lupeol was more effective than betulin.

CT phytochemicals; betulin; urolithiasis; urinary calculi; calcium; renal  
function; kidneys; triterpenoids; medicinal plants; non-wood forest  
products

L26 ANSWER 7 OF 9 CABA COPYRIGHT 2006 CABI on STN

ACCESSION NUMBER: 1999:159617 CABA  
 DOCUMENT NUMBER: 19990807661  
 TITLE: Targeting of piperine intercalated in  
 mannose-coated liposomes in experimental  
 leishmaniasis  
 AUTHOR: Barnini Raay; Swapna Medda; Sibabrata Mukhopadhyay;  
 Basu, M. K.; Raay, B.; Medda, S.; Mukhopadhyay, S.  
 CORPORATE SOURCE: Biomembrane Division, Indian Institute of Chemical  
 Biology, 4 Raja S.C. Mullick Road, Calcutta 700032,  
 India.  
 SOURCE: Indian Journal of Biochemistry & Biophysics, (1999)  
 Vol. 36, No. 4, pp. 248-251. 13 ref.  
 ISSN: 0301-1208  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 19991208  
 Last Updated on STN: 19991208

AB The leishmanicidal efficacy of piperine extracted from *Piper nigrum* and  
 incorporated into liposomes and mannose-coated liposomes was  
 tested in experimental visceral leishmaniasis in hamsters. Mannose  
 -coated liposomal piperine eliminated intracellular amastigotes of  
*Leishmania donovani* in splenic macrophages much more efficiently than  
 liposomal piperine or free piperine. At a dose equivalent to 6 mg/kg every  
 4 days and a total of 4 doses in 12 days, mannose-coated  
 liposomal piperine reduced the splenic parasite load by 90%, compared to  
 that achieved by liposomal piperine (77%) and free piperine (29%).  
 Histological examination of the spleen and liver function tests showed  
 that the toxicity of piperine was reduced when the mannosylated liposomal  
 formulation was administered.  
 CT leishmaniasis; liposomes; amastigotes; laboratory animals; experimental  
 infections; plant extracts; liver; liver  
 function; macrophages; spleen; visceral leishmaniasis; antiprotozoal  
 agents; drug formulations; mannose; adverse effects; drug  
 toxicity; drug delivery systems; parasites

L26 ANSWER 8 OF 9 CABA COPYRIGHT 2006 CABI on STN

ACCESSION NUMBER: 92:61114 CABA  
 DOCUMENT NUMBER: 19920309607  
 TITLE: Induction of rosmarinic acid accumulation in cell  
 suspension cultures of *Orthosiphon aristatus* after  
 treatment with yeast extract  
 AUTHOR: Sumaryono, W.; Proksch, P.; Hartmann, T.; Nimtz, M.;  
 Wray, V.  
 CORPORATE SOURCE: Institut fur Pharmazeutische Biologie der  
 Technischen Universitat Braunschweig, 3300  
 Braunschweig, Germany.  
 SOURCE: Phytochemistry, (1991) Vol. 30, No. 10, pp.  
 3267-3271. 21 ref.  
 ISSN: 0031-9422  
 DOCUMENT TYPE: Journal  
 LANGUAGE: English  
 ENTRY DATE: Entered STN: 19941101  
 Last Updated on STN: 19941101

AB The medicinal species *O. aristatus* is grown in Indonesia for export. The  
 tea prepared from its leafy shoots has diuretic properties and is used to  
 treat bacterial infections and inflammations of the  
 urinary system. Cell suspension cultures were shown to accumulate  
 the phenolic compound rosmarinic acid (RA) at concentrations of 1.0-2.0  
 [mu]mol/g FW. Addition of yeast extract (4-6 g/litre) to the liquid growth



media resulted in a large increase of RA accumulation in treated cells independent of the growth stage. The highest concentration of RA observed in treated cells (about 10  $\mu\text{mol/g FW}$ ) was usually reached 72-96 h after addition of yeast extract. When cells present in the stationary phase were treated with yeast extract a second phenolic was shown to accumulate which presumably originated by oxidative decarboxylation of RA. The induction of RA accumulation by yeast extract was due to de novo synthesis as shown by feeding experiments with  $^{14}\text{C}$ -tracers and by analysis of the activities of PAL and tyrosine aminotransferase (TAT) which are the key enzymes of RA biosynthesis. Following addition of yeast extract both enzyme activities showed a strong transient increase which preceded the peak of RA accumulation. Fractionation of yeast extract by acetone precipitation, ion exchange and gel permeation chromatography yielded two active fractions (elicitors A and B) capable of inducing RA accumulation. Both elicitors were shown to be carbohydrate polymers containing mainly **mannose**, glucose and to a lesser degree galactose. The elicitors are, thus, not identical with a glucan elicitor previously reported from yeast extract.

CT in vitro culture; metabolites; production; Phenolic compounds; Enzyme activity; Tissue culture; cells; Metabolism; phenols; Enzymes; aminotransferases; phenylalanine ammonia-lyase; **medicinal plants**

L26 ANSWER 9 OF 9 CABA COPYRIGHT 2006 CABI on STN

ACCESSION NUMBER: 91:111881 CABA

DOCUMENT NUMBER: 19910307871

TITLE: Glucosamine of the polysaccharide fractions from mistletoe and the Chinese parasol tree

AUTHOR: Murav'eva, D. A.; Popova, O. I.; Gasparyan, K. O.

CORPORATE SOURCE: Pyatigorsk Pharmaceutical Institute, Pyatigorsk, USSR.

SOURCE: Chemistry of Natural Compounds, (1990) Vol. 26, No. 6, pp. 705-706. translated from Russian original in Khimiya Prirodnykh Soedinenii (1990) 26 (6) 826-827. 4 ref.

ISSN: 0009-3130

DOCUMENT TYPE: Journal

LANGUAGE: English

ENTRY DATE: Entered STN: 19941101

Last Updated on STN: 19941101

AB Leaves of the Chinese parasol tree *Firmiana simplex* contained 4.2% water-soluble polysaccharides, 2.4% pectins and 4.3% hemicellulose; corresponding contents in leafy shoots of mistletoe, regardless of host, were around 4.4, 2.2 and 3.4%. Hydrolysates of soluble polysaccharides in mistletoes growing on pear, apple, willow and poplar trees contained similar amounts of glucosamine (0.54-0.73%), with much less (0.12-0.14%) in pectin hydrolysates. In *F. simplex*, glucosamine content in hydrolysate of soluble polysaccharide was highest (1.25%) for leaves, being 0.31-0.42% for seeds, inflorescences and fruit; corresponding values in pectin hydrolysates were 0.62% and 0.15-0.20%. The hydrolysates contained glucose, **mannose**, xylose, galactose and galacturonic acid.

CT composition; Polysaccharides; Parasitic weeds; Mistletoes; biology; pectins; hemicelluloses; leaves; glucosamine; Pears; host parasite relationships; Apples; Broadleaves; Plant composition; medicinal plants; weeds; ornamental plants; ornamental woody plants

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ACCESSION NUMBER: 84:43531 AGRICOLA  
DOCUMENT NUMBER: IND84025953  
TITLE: Bactrodesmium traversianum [Fungi, description, grown  
on *Salix* sp., distribution in British  
Columbia].  
AUTHOR(S): Hughes, S.J.; White, G.P.  
AVAILABILITY: DNAL (QK605.5.F8)  
SOURCE: Fungi Canadenses., Sept 1983 No. 259. 2 p ill  
Publisher: Ottawa : National Mycological Herbarium,  
Biosystematics Research Institute, Research Branch.  
DOCUMENT TYPE: Article  
FILE SEGMENT: Non-U.S. Imprint other than FAO  
LANGUAGE: English

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ACCESSION NUMBER: 80:49072 AGRICOLA  
DOCUMENT NUMBER: BRU80002135  
TITLE: INFECTIOUS ABORTION IN CATTLE THIRD REPORT.  
AUTHOR(S): RETTGER, L F; WHITE, G C; CHAPMAN, L  
AVAILABILITY: DNAL (100C76S)  
SOURCE: BULL CONN STORRS AGRIC EXP STN, 1921 p. 108  
NOTE: USDA EMPLOYEES ORDER FROM NAL LIBRARY. ALL OTHERS  
CONTACT VET-IN-CHARGE, VS-EPIC STAFF, HYATTSVILLE, MD-  
301-436-8418.  
DOCUMENT TYPE: Article  
LANGUAGE: English  
CT AGGLUTINABILITY OF CELLS; ALIMENTARY TRACT; ARTIFICIAL INSEMINATION;  
BOVINE; BRUCELLA ABORTUS; CASE HISTORY; COMPLEMENT FIXATION TEST;  
CONGENITAL; DAIRY PRODUCTS; ERADICATION; EXPERIMENTAL; HOST SPECIFICITY;  
HUSBANDRY PRACTICES; ORIGINAL EXPERIMENTAL WORK; REPRODUCTION; ROUTES OF  
ENTRY; SUBCUTANEOUS; TESTING FOR; URINARY SYSTEM; UROGENITAL  
TRACT

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=> d que 113

L5	3067	SEA FILE=WPIDS	ABB=ON	PLU=ON	MANNOSE
L6	254	SEA FILE=WPIDS	ABB=ON	PLU=ON	SALIX OR WILLOW (2A) BARK OR CRATAEVA
L7	5597	SEA FILE=WPIDS	ABB=ON	PLU=ON	POLLEN
L8	59843	SEA FILE=WPIDS	ABB=ON	PLU=ON	INFECTION?
L9	29890	SEA FILE=WPIDS	ABB=ON	PLU=ON	URIN?
L10	3	SEA FILE=WPIDS	ABB=ON	PLU=ON	L5 AND L6
L11	14	SEA FILE=WPIDS	ABB=ON	PLU=ON	L5 AND L7
L12	7	SEA FILE=WPIDS	ABB=ON	PLU=ON	L11 AND (L8 OR L9)
L13	9	SEA FILE=WPIDS	ABB=ON	PLU=ON	L10 OR L12

=> d que 120

L1	338	SEA FILE=WPIDS	ABB=ON	PLU=ON	WHITE G?/AU
L2	25	SEA FILE=WPIDS	ABB=ON	PLU=ON	ONEAL J?/AU
L3	21	SEA FILE=WPIDS	ABB=ON	PLU=ON	O NEAL J?/AU
L4	377	SEA FILE=WPIDS	ABB=ON	PLU=ON	(L1 OR L2 OR L3)
L5	3067	SEA FILE=WPIDS	ABB=ON	PLU=ON	MANNOSE
L6	254	SEA FILE=WPIDS	ABB=ON	PLU=ON	SALIX OR WILLOW (2A) BARK OR CRATAEVA
L8	59843	SEA FILE=WPIDS	ABB=ON	PLU=ON	INFECTION?
L9	29890	SEA FILE=WPIDS	ABB=ON	PLU=ON	URIN?
L20	11	SEA FILE=WPIDS	ABB=ON	PLU=ON	L4 AND (L5 OR L6 OR L8 OR L9)

=> d que 121

L1	338	SEA FILE=WPIDS	ABB=ON	PLU=ON	WHITE G?/AU
L2	25	SEA FILE=WPIDS	ABB=ON	PLU=ON	ONEAL J?/AU
L3	21	SEA FILE=WPIDS	ABB=ON	PLU=ON	O NEAL J?/AU
L4	377	SEA FILE=WPIDS	ABB=ON	PLU=ON	(L1 OR L2 OR L3)
L5	3067	SEA FILE=WPIDS	ABB=ON	PLU=ON	MANNOSE
L6	254	SEA FILE=WPIDS	ABB=ON	PLU=ON	SALIX OR WILLOW (2A) BARK OR. CRATAEVA
L7	5597	SEA FILE=WPIDS	ABB=ON	PLU=ON	POLLEN
L8	59843	SEA FILE=WPIDS	ABB=ON	PLU=ON	INFECTION?
L9	29890	SEA FILE=WPIDS	ABB=ON	PLU=ON	URIN?
L10	3	SEA FILE=WPIDS	ABB=ON	PLU=ON	L5 AND L6
L11	14	SEA FILE=WPIDS	ABB=ON	PLU=ON	L5 AND L7
L12	7	SEA FILE=WPIDS	ABB=ON	PLU=ON	L11 AND (L8 OR L9)
L13	9	SEA FILE=WPIDS	ABB=ON	PLU=ON	L10 OR L12
L14	2215	SEA FILE=WPIDS	ABB=ON	PLU=ON	MEDICINAL (S) PLANT?
L15	6	SEA FILE=WPIDS	ABB=ON	PLU=ON	L14 AND L8 AND L9

L16 2154 SEA FILE=WPIDS ABB=ON PLU=ON HERBAL (S) MEDICIN?  
 L17 6 SEA FILE=WPIDS ABB=ON PLU=ON L16 AND L8 AND L9  
 L18 11 SEA FILE=WPIDS ABB=ON PLU=ON L15 OR L17  
 L19 11 SEA FILE=WPIDS ABB=ON PLU=ON L18 NOT L13  
 L20 11 SEA FILE=WPIDS ABB=ON PLU=ON L4 AND (L5 OR L6 OR L8 OR L9)  
 L21 10 SEA FILE=WPIDS ABB=ON PLU=ON L20 NOT (L13 OR L19)

=> d .wp l13 1-9; d ibib ab l20 1-11; d ibib ab l21 1-10

L13 ANSWER 1 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

AN 2005-564516 [57] WPIDS

DNC C2005-170673

TI Regulating uptake of extracellular molecule by cell in subject, for treatment of condition e.g. viral **infection**, involves modulating functioning of structural or functional elements of cell's leverage mediated uptake mechanism.

DC B04 D16

IN SCHMIDT, O

PA (ADEL-N) ADELAIDE RES & INNOVATION PTY LTD

CYC 108

PI WO 2005074966 A1 20050818 (200557)\* EN 201

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT  
 KE LS LT LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG  
 ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE  
 DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG  
 KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ  
 OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG  
 US UZ VC VN YU ZA ZM ZW

ADT WO 2005074966 A1 WO 2005-AU150 20050207

PRAI AU 2004-900597 20040206

AB WO2005074966 A UPAB: 20050907

NOVELTY - Regulating the uptake of an extracellular molecule, preferably the uptake of a soluble adhesion molecule by a cell in a subject, involves modulating the functioning of any one or more structural or functional elements of the cell's leverage mediated uptake mechanism, preferably modulating the functioning of the molecule as a soluble adhesion molecule, or the interaction of the soluble adhesion molecule with one or more hinge molecules.

DETAILED DESCRIPTION - Regulating (M1) the uptake of an extracellular molecule by a cell in a subject, involves modulating the functioning of any one or more structural or functional elements of the cell's leverage mediated uptake mechanism, preferably regulating the uptake of a soluble adhesion molecule by a cell in a subject, involves modulating the functioning of the molecule as a soluble adhesion molecule, the interaction of the soluble adhesion molecule with one or more hinge molecules, the localization of the hinge molecule or the complex of the soluble adhesion molecule and hinge molecule proximally to both the surface membrane of the cell and one or more membrane anchored molecules (MARM), the interaction of the MARM with the soluble adhesion molecule, or the lateral clustering of the MARM relative to the hinge molecules.

INDEPENDENT CLAIMS are also included for:

(1) modulating (M2) cellular functional activity in a subject, which activity is induced and/or otherwise regulated by the assembly of an intracellular complex, involves modulating the functioning of any one or more structural or functional elements of the cell's leverage mediated uptake mechanism, where modulating the functioning of the elements regulates the functioning of the leverage mediated uptake mechanism;

(2) intracellular delivery (M3) of a molecule to a cell, involves

upregulating functioning of any one or more structural or functional elements of the cell's leverage mediated uptake mechanism, where upregulating the functioning of the elements upregulates the functioning of the leverage mediated uptake mechanism;

(3) down-regulating (M4) the microbial **infection** of a cell, involves down-regulating the functioning of any one or more structural or functional elements of the cell's leverage mediated uptake mechanism, where down-regulating the functioning of the elements down-regulates the functioning of the leverage mediated uptake mechanism;

(4) reducing (M5) pore-forming toxin induced cellular damage, involves down-regulating the functioning of any or more structural or functional element of the cell's leverage mediated uptake mechanism;

(5) modulating (M6) pore-forming toxin induced cellular damage in a subject, involves modulating the functioning of any or more structural or functional element of the cell's leverage mediated uptake mechanism, where down-regulating the functioning of the elements down-regulates the functioning of the leverage mediated uptake mechanism and upregulating the functioning of the elements upregulates the functioning of the leverage mediated uptake mechanism; and

(6) treatment and/or prophylaxis (M7) of condition in a subject, where the condition is characterized by the aberrant, unwanted or otherwise inappropriate cellular uptake an extracellular molecule or an intracellular complex, involves modulating the functioning of any one or more structural or functional elements of the cell's leverage mediated uptake mechanism.

ACTIVITY - Virucide; Antibacterial; Cytostatic.

No supporting data is given.

MECHANISM OF ACTION - Modulates cellular functional activity; Regulates uptake of extracellular molecule (claimed).

USE - (M1) is useful for regulation the uptake of an extracellular molecule by a cell in a subject, where the molecule is a drug, hormone, growth factor, antigen, modulator of intracellular signaling, immune regulator or pore forming toxin. (M1) is useful for the treatment and/or prophylaxis of a condition characterized by the aberrant, unwanted or otherwise inappropriate cellular uptake an extracellular molecule or an intracellular complex, such as viral **infection**, neoplasia or metastasis, bacterial **infection** and insect cellular resistance to *B.thuringiensis* (claimed).

Dwg.0/38

TECH

UPTX: 20050907

TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Method: In (M1), the extracellular molecule is a soluble adhesion molecule. The soluble adhesion molecule is proteinaceous or non-proteinaceous molecule, preferably multimeric molecule. The molecule is a drug, hormone, growth factor, antigen, modulator of intracellular signaling, immune regulator or pore forming toxin. The molecule is chosen from lectins, C-type lectins, galectins, pentraxins, C-reactive proteins, serum amyloid P, thio-ester proteins, a-macroglobulins, TEP, complement 3, serpin protease complex, serpin-thrombin, plasminogen activating protein, collectins, **mannose**-binding lectin, surfactant protein, Clq, ficollins, cysteine knot growth factors, endothelial growth factor (EGF), interleukins (IL), spaetzle, lipocalins, lactoferrin, asialoglycoproteins, advanced glycation end products, chitinase-like proteins, imaginal disc growth factor, bacterial enterotoxin:hemolysins, diphtheria toxin, cholera toxin, *Bacillus thuringiensis* toxin, limulin, CELIII, misteletoe lectin, ricin, wheat germ agglutinin and concavalin A. The MARM is chosen from mucins, glycoproteins, Clq, FcR, LRP, ClqR, CR3, lipocalin receptors, lactoferrin-receptor, ASGPR, galectin3, glycophorin, asialofetuin, aminopeptidase N, cadherin-like molecules and IL-2 receptor. The hinge molecule is an insect lipophorin-like protein, hexamerin-like

glycoprotein, lipocalin, pentraxin, apolipoprotein B100, apolipoprotein E or macroglobulin. In (M2), the cellular functional activity is cellular signaling, phagocytosis, attachment or detachment of the cell from an extracellular matrix or the formation of a cell-cell interaction. The cell-cell interactions are formed in the context of morphogenesis, tissue sculpturing, wound healing or cell division. The cellular functional activity is the inactivation of cellular functioning by destabilization of the actin cytoskeleton. The cellular functional activity is tip growth, which relates to angiogenesis, axon formation, **pollen** tube formation or rootlet formation. The cellular functional activity is intracellular protein secretion, endosome maturation, protein recycling or tissue compatibility. In (M3), the molecule is a biotic or abiotic compound such as a therapeutic or prophylactic drug or microorganism. The microorganism is a virion. The cell is a neoplastic cell and the drug is a pro-apoptotic drug. The cell is an infecting microorganism and the drug is a toxic compound. In (M4), the cell is an infecting microorganism and the drug is a toxic compound. In (M5) and (M6), the toxin is the endotoxin produced by *B.thuringiensis* and modulation is upregulation of cellular damage in insects. The modulation is down-regulation of pore-forming toxin induced cellular damage and the down-regulating achieved by exposure of the extracellular toxin to a mimic of the MARM or hinge molecule. In (M7), the condition is a viral **infection** and the modulation is down-regulating of the leverage mediated uptake mechanism for down-regulating the cellular uptake of the virus. The condition is a neoplasia or metastasis and the modulation is upregulation of the leverage mediated uptake mechanism for facilitating the delivery of a toxin. The condition is a bacterial **infection** and the modulating is upregulation of the leverage mediated uptake mechanism for facilitating the delivery of an antibiotic. The condition is insect cellular resistance to *B.thuringiensis* and the modulation is upregulation of the leverage mediated uptake mechanism for facilitating the entry of the *B.thuringiensis* to the insect cells.

L13 ANSWER 2 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 AN 2005-242392 [25] WPIDS  
 DNC C2005-077389  
 TI Producing antigenic response, involves contacting antigen-presenting cell with A1 adenosine receptor activating agent to increase antigenic response of antigen-presenting cell to antigen.  
 DC B04 D16  
 IN BORRON, P; WILSON, C N  
 PA (BORR-I) BORRON P; (WILS-I) WILSON C N; (ENDA-N) ENDACEA INC  
 CYC 108  
 PI WO 2005026318 A2 20050324 (200525)\* EN 56  
 RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE  
 LS LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW  
 W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE  
 DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG  
 KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ  
 OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG  
 US UZ VC VN YU ZA ZM ZW  
 US 2005075308 A1 20050407 (200525)  
 ADT WO 2005026318 A2 WO 2004-US24693 20040730; US 2005075308 A1 Provisional US  
 2003-491510P 20030731, US 2004-903933 20040730  
 PRAI US 2003-491510P 20030731; US 2004-903933 20040730  
 AB WO2005026318 A UPAB: 20060116  
 NOVELTY - Producing (M1) an antigenic response, involves contacting an antigen-presenting cell with an A1 adenosine receptor activating agent in an amount sufficient to increase the antigenic response of the antigen-presenting cell to the antigen.

DETAILED DESCRIPTION - Producing (M1) an antigenic response, involves:

- (a) contacting an antigen-presenting cell with an A1 adenosine receptor activating agent in an amount sufficient to increase the antigenic response of the antigen-presenting cell to the antigen;
- (b) administering to the subject, an A1 adenosine receptor agonist concurrently with an antigen in an amount sufficient to increase the antigenic response of the subject to the antigen, where the antigenic response is produced in a mammalian subject; or
- (c) transfecting or electroporating an antigen-presenting cell with a nucleotide sequence encoding an A1 adenosine receptor in a manner sufficient to increase the antigenic response of the antigen-presenting cell to antigen.

INDEPENDENT CLAIMS are also included for:

- (1) increasing (M2) a cytotoxic response induced by a cytotoxic cell, involves contacting the cytotoxic cell with an A1 adenosine receptor activating agent in an amount sufficient to increase the cytotoxic response of the cytotoxic cell;
- (2) enhancing (M3) A1 adenosine receptor signaling in an antigen-presenting cell, involves administering an activating agent to the antigen-presenting cell in an amount sufficient to enhance A1 adenosine receptor signaling in the antigen-presenting cell;
- (3) enhancing (M4) signaling between an antigen-presenting cell and an effector cell, involves administering an activating agent in an amount sufficient to enhance signaling between the antigen-presenting cell and the effector cell;
- (4) preventing (M5) desensitization of A1 adenosine receptor responses, involves administering to an antigen-presenting cell, a desensitizing agent in an amount sufficient to prevent desensitization of A1 adenosine receptor responses in the antigen-presenting cell, or transfecting or electroporating the antigen-presenting cell with a nucleotide sequence encoding a protein capable of preventing desensitization of A1 adenosine receptor responses;
- (5) a composition (C1) comprising an antigen and an activating agent;
- (6) a pharmaceutical composition (C2) comprising C1;
- (7) determining a subject's responsiveness to treatment for conditions associated with A1 adenosine receptor deficiency, involves determining A1 adenosine receptor expression, affinity or function on antigen-presenting cells;
- (8) imaging (M6) antigen-presenting cells in vivo in a subject, involves:
  - (a) obtaining a sample of antigen-presenting cells from a subject, labeling the antigen-presenting cells with a radiolabeled A1 adenosine receptor ligand, nucleotide sequence encoding the A1 adenosine receptor, and then administering the labeled antigen-presenting cells to the subject in an amount effective to provide a radioimage; or
  - (b) obtaining a sample of antigen-presenting cells from a subject, and contacting the antigen-presenting cell with a biosensor that recognizes a specific target on the antigen-presenting cell, with the proviso that the biosensor is not a radiolabeled biosensor; and
- (9) a diagnostic kit for determining a subject's responsiveness to treatment for conditions associated with A1 adenosine receptor deficiency, comprising at least one reagent for determining A1 adenosine receptor expression, affinity, or function on antigen-presenting cells of the subject, and printed instructions for assessing the subject's responsiveness to treatment for conditions associated with A1 adenosine receptor deficiency, where at least one reagent and the printed instructions are packaged together in a container.

ACTIVITY - CNS-Gen.; Antimicrobial; Immunosuppressive; Muscular-Gen.; Neuroprotective; Antiinflammatory; Vasotropic; Antidiabetic; Cytostatic;

Antiasthmatic; Antiallergic; Antiarteriosclerotic.

No supporting data is given.

MECHANISM OF ACTION - Immunestimulator.

USE - (M1) is useful for producing an antigenic response, where the method of producing the antigenic response is performed in combination with known methods of treatment of conditions chosen from immunodeficiency disorders, central nervous system (CNS) disorders, infectious diseases, autoimmune diseases, myasthenia gravis, Crohn's disease, regional enteritis, vasculitis, diabetes mellitus, tumors, cancer, substance abuse, multiple sclerosis, asthma, contact allergy, transplant rejection and atherosclerosis. C1 is useful for immunizing a mammal against an antigen, which involves administering C1. C1 is useful for treating conditions as mentioned above, which involves administering to subject, C1 in an amount sufficient to treat the condition, where the condition is prostate cancer (claimed).

Dwg.0/0

TECH

UPTX: 20050419

TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Method: (M1) for producing an antigenic response, involves contacting a dendritic cell with an A1 adenosine receptor agonist in an amount sufficient to increase the antigenic response of the dendritic cell to the antigen. In (a) of (M1), the antigenic response is an immune response, and is an adaptive immune response or an innate immune response. The increase in immune response is a phenotypic or genotypic increase in responsiveness to an antigen, or is the production of higher antibody titers, increase in antibody affinity generation of cytotoxic cells or increase in tolerogenic response. The antigen-presenting cell is chosen from monocytes, macrophages, dendritic cells, Langerhans cells, lymphocytes, hematopoietic stem cells, peripheral blood stem cells, peripheral blood mononuclear cells, B cells, veiled cells, interdigitating and follicular cells, splenocytes, thymocytes, microglia, Kupffer cells, endothelial cells, fibroblasts and eosinophils. The antigen is a live microorganism, non-living compound, composition, or is an antibody-inducing determinant. The antigen is chosen from peptide, protein, lipid, carbohydrate, nucleic acid, mucin, proteoglycan and their combinations and derivatives. The antigen comprises interleukin (IL)-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-9, IL-10, IL-11, IL-12, IL-13, IL-15, IL-16, IL-17, prostaglandins, thromboxane, leukotrienes, platelet activating factor (PAF), lipid A, phospholipase A2, endotoxins, Staphylococcal enterotoxin B, type I interferon, type II interferon, TNF-alpha, transforming growth factor-beta (TGF-beta), lymphokines, lymphotoxin migration inhibition factor, granulocyte-macrophage colony-stimulating factor (GM-CSF), monocyte-macrophage CSF, granulocyte CSF, vascular epithelial growth factor (VEGF), angiogenin, TGF-alpha, heat shock proteins (HSPs), carbohydrate moieties of blood groups, Rh factors, fibroblast growth factor (FGF), eosinophil (EOS) cationic protein, EOS granule proteins, regulated on activation, normal T cell expressed and secreted (RANTES), nucleotides, nucleosides, DNA, RNA, mRNA, MART, MAGE, BAGE, mutant p53, tyrosinase, Azidothymidine (AZT), angiostatin, endostatin, tumor, cancer, viral **infections**, bacterial **infections**, fungal **infections**, atypical bacterial **infections**, parasitic **infections**, protozoal **infections**, self-antigens, alloantigens, transplant antigens, graft antigens, oncofetal antigens, tumor associated mucins, tumor-derived peptides, tumor cell lysates, toxins, dead cells, necrotic cells, lipopolysaccharide (LPS), exotoxin, enterotoxin, 1,3 beta glucan, peptidoglycan, lipoteichoic acid, **mannose**, flagellin, pilin, glycolipids, zymosan, cytokines, chemokines, immune complexes, haptens, alcohol, drugs, monocyte chemoattractant protein (MCP), MCP-1, MCP-3, MCP-4, MIF, HMGB1, MIP-1alpha, MIP-1beta, MIP-3alpha, MIP-5/human cc cytokine-2 (HCC2), CD40 ligand (CD40L), TNF-related activation induced



cytokine (TRANCE), Flt 3 ligand (FL), c-kit, C5a, complement, stem cell factor (SCF), hepatocyte growth factor (HGF), macrophage-derived chemokines (MDC), stromal cell derived factor-1alpha (SDF-1alpha), prions, bovine spongiform encephalomyelitis protein (BSE), prostate specific antigen (PSA), prostate alkaline phosphatase (PAP), amyloid precursor protein (APP), amyloid beta (Abeta), tau, xenoantigens, superantigens, ovalbumin, ragweed, house dust mite, plant **pollens**, plant molecules, insect toxins, chemicals, A1 adenosine receptors, P2X purinoceptors, B cell receptors, T cell receptors, antibodies and their combinations. The antigen-presenting cell expresses at least one A1 adenosine receptor. The contacting step is carried out in vitro or in vivo. (M1) further involves priming the antigen-presenting cell by contacting the antigen-presenting cell with a priming agent in an amount sufficient to prime the antigen-presenting cell, and activating the antigen-presenting cell by contacting the antigen-presenting cell with an activating agent in an amount sufficient to induce the antigen-presenting cells to mediate an increase in immune response to an antigen. In (b) of (M1), the step of administering the A1 adenosine receptor agonist is carried out simultaneously or sequentially with the step of administering the antigen. In (c) of (M1), the nucleotide sequence is a cDNA encoding a human A1 adenosine receptor. In (M2), the activating agent is chosen from A1 adenosine receptor agonists, cisplatin, dexamethasone, daunorubicin, doxorubicin, mitoxantrone, carbamazepine, adenosine receptor antagonists, nucleotide sequences encoding the A1 adenosine receptor, allosteric enhancers, and protein kinase inhibitors. The cytotoxic response is an increase in the biological responses chosen from tumoricidal activity, tumoristatic activity, phagocytosis, lysis and production of biological response modifiers, compared to the cytotoxic response in the absence of contacting the cytotoxic cell with an activating agent. The cytotoxic cell is chosen from natural killer cells, cytotoxic lymphocytes, lymphokine activated killer cells, macrophages, Kupffer cells, microglia, dendritic cells, antibody secreting cells and cells secreting other effector molecules. The cytotoxic cell expresses at least one A1 adenosine receptor. (M2) further involves priming the cytotoxic cell by contacting the cytotoxic cell with a priming agent in an amount sufficient to prime the cytotoxic cell, and activating the cytotoxic cell by contacting the cytotoxic cell with an activating agent in an amount sufficient to induce the cytotoxic cell to mediate an increase in biological responses chosen from tumoricidal activity, tumoristatic activity, phagocytosis, lysis and production of biological response modifiers. In (M3), the step of enhancing A1 adenosine receptor signaling, involves correcting an A1 adenosine receptor deficiency in the antigen-presenting cell and further involves administering an activating agent to the antigen-presenting cell in an amount sufficient to increase the number of A1 adenosine receptors on the antigen-presenting cell plasma membrane. The activating agent is chosen from A1 adenosine receptor agonists, cisplatin, dexamethasone, daunorubicin, doxorubicin, mitoxantrone, carbamazepine, adenosine receptor antagonists, nucleotide sequence encoding the A1 adenosine receptor, subjecting the cells to ischemic conditions, allosteric enhancers and protein kinase inhibitors. The step of enhancing A1 adenosine receptor signaling, involves correcting an A1 adenosine receptor deficiency in the antigen-presenting cell and further involves genetically altering A1 adenosine receptor expression in the antigen-presenting cell or chemically altering A1 adenosine receptor expression in the antigen-presenting cell. In (M4), the effector cell is chosen from monocytes, macrophages, lymphocytes, B cells, T cells, natural killer cells, mast cells, basophils, eosinophils, plasma cells, microglia, Kupffer cells, granulocytes, fibroblasts, and endothelial cells. The activating agent is administered in the presence or absence of an antigen. In (M5), the desensitizing agent is chosen from adenosine deaminase, allosteric

enhancers, and protein kinase inhibitors. The nucleotide sequence encoding a protein capable of preventing desensitization of A1 adenosine receptor responses leads to an increased expression of spinophilin, alkaline phosphatase, protein phosphatase 1 (PP1), or protein phosphatase 2A (PP2A). The (a) of (M6) further involves priming the antigen-presenting cells by contact with a priming agent in an amount sufficient to prime the antigen-presenting cells. In (b) of (M6), the specific target is an A1 adenosine receptor or is an extracellular domain of an A1 adenosine receptor.

Preferred Composition: C1 further comprises an immunomodulator, a priming agent, carrier and adjuvant. C1 is lyophilized, and is an immunogenic composition. The carrier is an aqueous carrier or a solid carrier.

L13 ANSWER 3 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

AN 2004-766883 [75] WPIDS

CR 2004-766856 [75]; 2005-173108 [18]

DNC C2004-268974

TI New isolated nucleic acid molecule comprises a sequence encoding Stress-Related Protein (SRP), useful for producing transformed plants with altered metabolic activity resulting in increased tolerance or resistance to environmental stress.

DC C06 D16

IN CHARDONNENS, A; CHEN, R; MCKERSIE, B; PUZIO, P; SARRIA-MILLAN, R; SHIRLEY, A; WANG, X

PA (BADI) BASF PLANT SCI GMBH

CYC 108

PI WO 2004092398 A2 20041028 (200475)\* EN 911

RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE  
LS LU MC MW MZ NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE  
DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG  
KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ  
OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG  
US UZ VC VN YU ZA ZM ZW

ADT WO 2004092398 A2 WO 2004-US11888 20040415

PRAI EP 2003-22225 20030930; EP 2003-8080 20030415;

EP 2003-39728 20030502; EP 2003-16672 20030801

AB WO2004092398 A UPAB: 20050316

NOVELTY - An isolated nucleic acid molecule comprises a nucleic acid molecule encoding a Stress-Related Protein (SRP), is new.

DETAILED DESCRIPTION - An isolated nucleic acid molecule comprises a nucleic acid molecule selected from:

(a) nucleic acid molecule encoding of the polypeptide given in the specification or its fragment, which confers an altered metabolic activity in an organism or its part;

(b) nucleic acid molecule comprising of the nucleic acid molecule given in the specification;

(c) nucleic acid molecule whose sequence can be deduced from a polypeptide sequence encoded by a nucleic acid molecule of (a) or (b) as a result of the degeneracy of the genetic code and confers an altered metabolic activity in an organism or its part;

(d) nucleic acid molecule which encodes a polypeptide which has at least 50% identity with the amino acid sequence of the polypeptide encoded by the nucleic acid molecule of (a) to (c) and confers an altered metabolic activity in an organism or its part;

(e) nucleic acid molecule which hybridizes with a nucleic acid molecule of (a) to (c) and confers an altered metabolic activity in an organism or its part;

(f) nucleic acid molecule which encompasses a nucleic acid molecule which is obtained by amplifying nucleic acid molecules from a cDNA library

or a genomic library using the primers given in the specification and confers an altered metabolic activity in an organism or its part;

(g) nucleic acid molecule encoding a polypeptide which is isolated with the aid of monoclonal antibodies against a polypeptide encoded by one of the nucleic acid molecules of (a) to (f) and confers an altered metabolic activity in an organism or its part;

(h) nucleic acid molecule encoding a polypeptide comprising the consensus sequence given in the specification and confers an altered metabolic activity in an organism or its part; or

(i) nucleic acid molecule which is obtainable by screening a suitable nucleic acid library with a probe comprising one of the sequences of the nucleic acid molecule of (a) to (h) or with a fragment having at least 15 nt, preferably 20 nt to 500 nt of the nucleic acid molecule in (a) to (h) and confers altered metabolic activity in an organism or its part, where the nucleic acid molecule distinguishes over the sequence given in the specification by one or more nucleotides.

INDEPENDENT CLAIMS are also included for:

(1) a transformed plant cell with altered metabolic activity compared to a corresponding non-transformed wild type plant cell, where the metabolic activity is altered by transformation with a SRP coding nucleic acid and results in increased tolerance and/or resistance to an environmental stress as compared to a corresponding non-transformed wild type plant cell;

(2) a transgenic plant generated from the plant cell above and which is a monocot or dicot plant, or a gymnosperm plant;

(3) a seed produced by a transgenic plant above, where the seed is genetically homozygous for a transgene conferring altered metabolic activity resulting in an increased tolerance to environmental stress as compared to a corresponding non-transformed wild type plant;

(4) a nucleic acid construct which confers the expression of the nucleic acid molecule above comprising one or more regulatory elements, where expression of the SRP coding nucleic acid in a host cell results in altered metabolic activity resulting in increased tolerance to environmental stress as compared to a corresponding non-transformed wild type host cell;

(5) a vector comprising the nucleic acid molecule above or the nucleic acid construct of (4);

(6) a host cell, which has been transformed stably or transiently with the vector of (5), the nucleic acid molecules above, or the nucleic acid construct of (4);

(7) an isolated Stress Related Protein (SRP) selected from the amino acid sequences fully given in the specification and/or its homologues;

(8) a method of producing a transgenic plant with altered metabolic activity compared to a corresponding non-transformed wild type plant cell;

(9) modifying stress tolerance of a plant;

(10) detecting environmental stress in plant cells or plants;

(11) screening plant cells or plants for increased tolerance and/or resistance to environmental stress;

(12) breeding plant cells or plants towards increased tolerance and/or resistance to environmental stress;

(13) a plant cell comprising the nucleic acid construct of (4) or the vector of (5);

(14) a plant comprising the cell of (15);

(15) an isolated nucleic acid comprising a polynucleotide selected from: (a) a nucleic acid sequence fully given in the specification; (b) a polynucleotide encoding a polypeptide fully given in the specification; (c) a polynucleotide having at least 70% sequence identity with a (a), where expression of the polynucleotide confers increased tolerance to one or more abiotic stress in a plant as compared to a corresponding non-transformed plant; or (d) a polynucleotide hybridizing to (a), where

expression of the polynucleotide confers increased tolerance to one or more abiotic stress in a plant as compared to a corresponding non-transformed plant;

(16) a vector comprising the isolated nucleic acid of (17);

(17) a plant stably transformed with the isolated nucleic acid of (17);

(18) a plant stably transformed with the vector of (19);

(19) a seed of the plant of (20), where the seed comprises the isolated nucleic acid of (17) or the vector of (18);

(20) increasing tolerance of a plant to at least one abiotic stress;

(21) a plant transformed with the nucleic acids above; and

(22) a seed of the plant of (23).

USE - The SRP encoding nucleic acids or its homologues are useful for preparing a plant cell with increased environmental stress tolerance. The altered metabolic activity and/or a SRP encoding nucleic acids or its homologues are useful as markers for selection of plants or plant cells with increased tolerance to environmental stress, or for detection of stress in plants or plant cells (all claimed). The nucleic acids are useful for producing transformed plants with altered metabolic activity resulting in increased tolerance and/or resistance to an environmental stress as compared to a corresponding non-transformed wild-type plant cell.

Dwg. 0/2

TECH

UPTX: 20041122

TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Plant/Plant Cell: In the plant cell above, the metabolic activity is altered concerning one or more metabolites selected from 2,3-dimethyl-5-phytylquinol, 2-hydroxy-palmitic acid, 3,4-dihydroxyphenyl-alanine (= dopa), 3-hydroxy-palmitic acid, 5-oxoproline, alanine, alpha linolenic acid (c18:3 (c9, c12, c15)), alpha-tocopherol, aminoadipic acid, anhydroglucose, arginine, aspartic acid, beta-apo-8' carotenal, beta-carotene, beta-sitosterol, beta-tocopherol, (delta-7-cis,10-cis)-hexadecadienic acid, hexadecatrienic acid, margaric acid, delta-15-cis-tetracosenic acid, ferulic acid, campesterol, cerotic acid (c26:0), citrulline, cryptoxanthine, eicosenoic acid (20:1), fructose, fumarate, galactose, gamma-aminobutyric acid, gamma-tocopherol, gluconic acid, glucose, glutamic acid, glutamine, glycerate, glyceraldehyde, glycerol, glycerol-3-phosphate, glycine, homoserine, inositol, isoleucine, iso-maltose, isopentenyl pyrophosphate, leucine, lignoceric acid (c24:0), linoleic acid (c18:2 (c9, c12)), luteine, lycopene, malate, mannose, methionine, methylgalactofuranoside, methylgalactopyranoside, palmitic acid (c16:0), phenylalanine, phosphate, proline, putrescine, pyruvate, raffinose, ribonic acid, serine, shikimate, sinapine acid, stearic acid (c18:0), succinate, sucrose, threonine, triacontanoic acid, tryptophane, tyrosine, ubiquinone, udp-glucose, valine, or zeaxanthine. The metabolic activity is altered by transformation with a SRP coding nucleic acid selected from the nucleic acid molecules above. Environmental stress is selected from salinity, drought, temperature, metal, chemical, pathogenic, or oxidative stresses, or its combinations. The transgenic plant cell is derived from a monocotyledonous plant or a dicotyledonous plant selected from maize, wheat, rye, oat, triticale, rice, barley, soybean, peanut, cotton, rapeseed, canola, manihot, pepper, sunflower, flax, borage, safflower, linseed, primrose, turnip rape, tagetes, solanaceous plants, potato, tobacco, eggplant, tomato, Vicia species, pea, alfalfa, coffee, cacao, tea, Salix species, oil palm, coconut, perennial grass, forage crops, or Arabidopsis thaliana. The transgenic plant cell is also derived from a gymnosperm plant. Preferably, the plant is spruce, pine, or fir. Preferred Stress Related Protein: The Stress Related Protein is selected from yeast, preferably Saccharomyces cerevisiae, or Escherichia colie, Brassica napus, Glycine max, or Oryza sativa.

Preferred Method: Producing a transgenic plant with altered metabolic activity compared to a corresponding non transformed wild type plant cell comprises transforming a plant cell with an expression vector above, and generating from the plant cell a transgenic plant with an increased tolerance to environmental stress as compared to a corresponding non-transformed wild type plant. The SRP coding nucleic acid is selected from nucleic acid sequences fully given in the specification and/or its homologues. The SRP coding nucleic acid is at least about 50% homologous to the nucleic acids fully given in the specification. Modifying stress tolerance of a plant comprises modifying the level of expression of an SRP in the plant. An expression vector is used in the method above. The stress tolerance is decreased. The plant is transgenic. It is transformed with an inducible promoter that directs expression of the SRP. The promoter is tissue specific or developmentally regulated. The SRP expression is modified by administration of a targeting nucleic sequence complementary to the regulatory region of the SRP encoding nucleic acid and/or by a transcription factor and/or by a zinc finger protein. Detecting environmental stress in plant cells or plants comprises screening the plant cells for altered metabolic activity as compared to non-stress conditions. Screening plant cells or plants for increased tolerance and/or resistance to environmental stress comprises screening the plant cells under stress conditions for altered metabolic activity as compared to non-stress conditions. Breeding plant cells or plants towards increased tolerance and/or resistance to environmental stress comprises screening the plant cells under stress conditions for altered metabolic activity as compared to non-stress conditions and selecting those with increased tolerance and/or resistance to environmental stress. The altered metabolic activity is by transformation with SRP coding nucleic acid. It is transformation with one or more SRP coding nucleic acids fully given in the specification and/or its homologues. Increasing tolerance of a plant to at least one abiotic stress comprises transforming a plant with the isolated nucleic acids above or the vectors above.

L13 ANSWER 4 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 AN 2004-766856 [75] WPIDS  
 CR 2004-766883 [75]; 2005-173108 [18]  
 DNC C2004-268947  
 TI New transformed plant cell with altered metabolic activity compared to a corresponding non transformed wild type plant cell, useful for producing, screening and breeding plants with increased tolerance to environmental stress.  
 DC C06 D16 P13  
 IN CHARDONNENS, A; CHEN, R; PUENTE, P; PUZIO, P  
 PA (BADI) BASF PLANT SCI GMBH  
 CYC 109  
 PI WO 2004092349 A2 20041028 (200475)\* EN 607  
 RW: AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE  
 LS LU MC MW MZ NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW  
 W: AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE  
 DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG  
 KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ  
 OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG  
 US UZ VC VN YU ZA ZM ZW  
 EP 1615998 A2 20060118 (200606) EN  
 R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HR HU IE IT LI LT LU  
 LV MC MK NL PL PT RO SE SI SK TR  
 ADT WO 2004092349 A2 WO 2004-US11887 20040415; EP 1615998 A2 EP 2004-759579  
 20040415, WO 2004-US11887 20040415  
 FDT EP 1615998 A2 Based on WO 2004092349  
 PRAI EP 2003-22226 20030930; EP 2003-8079 20030415;

EP 2003-16671 20030801

AB WO2004092349 A UPAB: 20060124

NOVELTY - A transformed plant cell with altered metabolic activity compared to a corresponding non transformed wild type plant cell, where the metabolic activity is altered by an inactivated or down-regulated gene and results in increased tolerance and/or resistance to an environmental stress as compared to a corresponding non-transformed wild type plant cell, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following:

(1) a transformed plant generated from the plant cell, which is a monocot or a dicot plant;

(2) a seed produced by the transformed plant, where the seed is at least genetically heterozygous for a gene, that when inactivated or down-regulated confers increased tolerance to environmental stress as compared to a wild type plant;

(3) a method of producing a transformed plant with altered metabolic activity compared to a corresponding non transformed wild type plant cell by inactivation or down-regulation of a gene in the transformed plant resulting in increased tolerance and/or resistance to environmental stress as compared to a corresponding non-transformed wild type plant;

(4) a method of inducing increased tolerance and/or resistance to environmental stress as compared to a corresponding non-transformed wild type plant in the plant cell or plant by altering metabolic activity compared to a corresponding non transformed wild type plant cell by inactivation or down-regulation of one or more genes encoded by one or more nucleic acids;

(5) a plant expression cassette comprising a nucleic acid construct, which when expressed allows inactivation or down-regulation of one or more genes encoded by one or more nucleic acids;

(6) a method of detecting environmental stress in plant cells or plants;

(7) a method of screening plant cells or plants for increased tolerance and/or resistance to environmental stress;

(8) a method of breeding plant cells or plants towards increased tolerance and/or resistance to environmental stress;

(9) a transformed plant cell with an inactivated or down-regulated gene encoded by a nucleic acid sequence selected from any of those given in the specification;

(10) an isolated nucleic acid molecule, which, as a result of the degeneracy of the genetic code, can be derived from a polypeptide sequences given in the specification and having biological activity;

(11) an isolated polypeptide encoded by the nucleic acid molecule;

(12) an antibody, which specifically binds to the polypeptide;

(13) a transformed plant cell where the increased tolerance and/or resistance to an environmental stress is conferred by one or more inactivated or down-regulated genes encoded by one or more nucleic acid sequences cited above; and

(14) a plant comprising the cell.

USE - The transformed plant cell is useful for producing, screening and breeding plants with increased tolerance to environmental stress, and for detecting stress in cells or plants.

Dwg.0/0

TECH UPTX: 20041122

TECHNOLOGY FOCUS - BIOTECHNOLOGY - Preferred Cell: The transformed plant cell comprises a metabolic activity that is altered concerning one or more metabolites selected from 2,3-dimethyl-5-phytylquinol, 2-hydroxy-palmitic acid, 3,4 dihydroxyphenylalaninedopa, 3-hydroxy-palmitic acid, 5-oxoproline, alanine, alpha linolenic acid, alpha-tocopherol, aminoadipic acid, anhydroglucose, arginine, aspartic acid, beta-apo-81 carotenal,

beta-carotene, beta-sitosterol, beta-tocopherol, (delta-7-cis,10-cis)-hexadecadienic acid, hexadecatrienic acid, margaric acid, delta-15-cis-tetracosenic acid, ferulic acid, campesterol, cerotic acid (c26:0), citrulline, cryptoxanthine, eicosenoic acid (20:1), fructose, fumarate, galactose, gamma-aminobutyric acid, gamma-tocopherol, gluconic acid, glucose, glutamic acid, glutamine, glycerate, glycerinaldehyde, glycerol, glycerol-3-phosphate, glycine, homoserine, inositol, isoleucine, iso-maltose, isopentenyl pyrophosphate, leucine, lignoceric acid (c24:0), linoleic acid (c18:2 (c9, c12)), luteine, lycopene, malate, **mannose**, methionine, methylgalactofuranoside, methylgalactopyranoside, methylgalactopyranoside, palmitic acid (c16:0), phenylalanine, phosphate, proline, putrescine, pyruvate, raffinose, ribonic acid, serine, shikimate, sinapine acid, stearic acid (c18:0), succinate, sucrose, threonine, triacontanoic acid, tryptophane, tyrosine, ubiquinone, udp-glucose, valine, and zeaxanthine. The metabolic activity is altered by one or more inactivated or down-regulated genes encoded by one or more nucleic acid sequences. The plant is selected from maize, wheat, rye, oat, triticale, rice, barley, soybean, peanut, cotton, rapeseed, canola, manihot, pepper, sunflower, flax, borage, safflower, linseed, primrose, rapeseed, turnip rape, tagetes, solanaceous plants, potato, tobacco, eggplant, tomato, Vicia species, pea, alfalfa, coffee, cacao, tea, **Salix** species, oil palm, coconut, perennial grass, forage crops and Arabidopsis thaliana.

The environmental stress is selected from salinity, drought, temperature, metal, chemical, pathogenic and oxidative stresses, or their combinations. The transformed plant cell is derived from a monocotyledonous or dicotyledonous plant. It is derived from a gymnosperm plant. The plant is selected from spruce, pine and fir.

Preferred Method: In inducing increased tolerance and/or resistance to environmental stress as compared to a corresponding non-transformed wild type plant in a plant cell, or plant, the inactivation or down-regulation of the gene is achieved by double-stranded RNA interference (dsRNAi), introduction of an antisense nucleic acid, a ribozyme, an antisense nucleic acid combined with a ribozyme, a nucleic acid encoding a co-suppressor, a nucleic acid encoding a dominant negative protein, DNA- or RNA- or protein-binding factors targeting said gene or -RNA or -proteins, RNA degradation inducing viral nucleic acids and expression systems, systems for inducing a homolog recombination of the genes, mutations in the genes or their combinations.

L13 ANSWER 5 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

AN 2004-570523 [55] WPIDS

DNC C2004-208289

TI Use of D-**mannose** for maintenance of **urinary** tract health in the face of **infection** e.g. E. Coli **infection**

DC B03 B04

IN ONEAL, J; WHITE, G

PA (ONEA-I) ONEAL J; (WHIT-I) WHITE G

CYC 1

PI US 2004147459 A1 20040729 (200455)\* 6

ADT US 2004147459 A1 Provisional US 2002-420696P 20021023, US 2003-691423 20031022

PRAI US 2002-420696P 20021023; US 2003-691423 20031022

AB US2004147459 A UPAB: 20040826

NOVELTY - Maintenance of **urinary** tract health in the face of **infection** involves administering a dosage of 1 - 2 teaspoons of D-**mannose** three times a day with meals for 1 - 2 weeks or until the symptoms subside.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a

composition comprising a therapeutic dosage of D-mannose and a therapeutic dosage of at least one of an extract of *Crataeva nurvala*, *willow bark* or *pollen* extract simultaneously with D-mannose.

ACTIVITY - Uropathic; Antimicrobial.

MECHANISM OF ACTION - E. coli urethral epithelial cell attachment inhibitor.

USE - For maintaining **urinary** tract health in the face of **infection**; in combination with a capsule containing herbs that affect the **urinary** tract; and for dealing with a **urinary tract infection** (all claimed). The **urinary tract infection** include E. coli **infection**.

ADVANTAGE - The use of D-mannose in the maintenance of **urinary** tract health in the face of **infection** and in the treatment of **urinary tract infection** preferentially provides attachment of E. coli fimbriae to the administered D-mannose present in the **urine**, rather than attachment to D-mannose in the epithelial cells of the **urinary** tract. This results in the E. coli bacteria surrounded by the molecules of D-mannose and promotes their natural elimination by mechanical and not pharmacological action. The few remaining bacteria can then be better handled by the body's natural defenses, the white blood cells. Mannose can not be broken down in the body, and thus is safe for diabetics, pregnant women and the elderly, and is virtually free from the risk of overdose. The method does not involve the use of antibiotics and hence avoids the side effects and resistant strain development associated with them. The administration regimen provides a quantity of mannose sufficient to remove a majority of E/ coli in the **urinary** tract, while improving the ease of use and compliance. The dosages are effective in doctor-run trials.

Dwg.0/3

TECH

UPTX: 20040826

TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Method: D-Mannose is administered in a capsule containing herbs that affect the **urinary** tract. The method further involves administering at least one of an extract of *Crataeva nurvala*, *willow bark* or *pollen* extract simultaneously with D-mannose.

L13 ANSWER 6 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

AN 2004-070543 [07] WPIDS

DNC C2004-029123

TI Production of fermented hydrolyzed medium for treating e.g. infectious disease, by mixing small pieces of solid food ingredient with liquid ingredient, adding sugar and fermenting until mixture reaches preset acidity.

DC B04 C03 D13 D16 D21

IN SOBOL, C V; SOBOL, Y T

PA (SOBO-I) SOBOL C V; (SOBO-I) SOBOL Y T; (TECH-N) TECHNOLOGY COMMERCIALIZATION CORP

CYC 100

PI US 2003235559 A1 20031225 (200407)\* 9

WO 2004000038 A1 20031231 (200407) EN

RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS

LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK

DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR

KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT

RO RU SD SE SG SK SL TJ TM TN TR TT TZ UA UG UZ VN YU ZA ZM ZW

AU 2003236536 A1 20040106 (200447)



US 6953574 B2 20051011 (200567)  
ADT US 2003235559 A1 US 2002-178447 20020621; WO 2004000038 A1 WO 2003-US18831  
20030613; AU 2003236536 A1 AU 2003-236536 20030613; US 6953574 B2 US  
2002-178447 20020621  
FDT AU 2003236536 A1 Based on WO 2004000038  
PRAI US 2002-178447 20020621  
AB US2003235559 A UPAB: 20040128

NOVELTY - Production of a fermented hydrolyzed medium, comprising mixing small pieces of solid food ingredient (70-5 %, by weight) with the biocompatible liquid ingredient (10-90 %, by weight), adding sugar (0.1-30 % by weight) to the mixture and fermenting the mixture at 35-58 deg. C until the mixture reaches an acidity of 300-900 deg. T and obtaining high acidity medium with high concentration of microorganism and their metabolic products, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) treating a medical condition comprising producing a hydrolyzed medium with high acidity of at least 300 deg. T, containing non-pathogenic microorganism and their highly concentrated metabolic products, by:

(a) fermenting a mixture of biocompatible liquid ingredient and sugar ingredient for 3-14 days at 10-58 deg. C; and

(b) applying the medium by oral, transcutaneous, inhalation, rectal, vaginal, nasal, topical, intravenous after removal of microorganism and intraperitoneal; and

(2) increasing skin elasticity, promoting skin regeneration, reducing hems, scars and wrinkles, and healing burnt skin which involves producing the medium as above and applying on the skin.

ACTIVITY - Virucide; Fungicide; Protozoacide; Antibacterial; Cytostatic; Cardiovascular-Gen.; Nephrotropic; Hepatotropic; Gastrointestinal-Gen.; Neuroprotective; Immunosuppressive; Antidiabetic; Anti-HIV; Tuberculostatic; Antiinflammatory; Respiratory-Gen.; Nootropic; Endocrine-Gen.

No biological data is given.

MECHANISM OF ACTION - None given.

USE - For treating and preventing viral, fungi, protozoal or bacterial **infection**, cancer, cardiovascular disease, kidney or liver condition, digestion disease, neurodegenerative disease, transplanted organ, autoimmune disease, diabetic condition, excessive radiation condition, HIV/AIDS, tuberculosis, hepatitis, herpes, influenza, poliomyelitis virus, lung disease, Alzheimer's disease or dementia, disease of sexual organs and abnormality (claimed), and as a feed supplement for prevention and treatment of **infection** disease, for growth promotion to improve feed conversion and to increase the yield of useful products such as milk and eggs, used in cosmetics industry for improving skin and in food industry for production of dairy products, to accelerate cheese ripening and maturation, and for improving the cultivation and long-term storage preservation of viable Lactobacilli, acetic, propionic and bifidobacteria in their most active state.

ADVANTAGE - The medium has physiologically beneficial effects and therapeutic activity against various diseases, including several life-threatening condition. The fermented medium allows the microorganism to remain alive in active state, so that better conditions are created for attachment to the appropriate tissue of the host. The medium has high acidity and promotes higher vitality of the microorganisms. The medium is produced using the natural sources such as fruits and vegetables and does not require heavy food processing or heat. The medium contains live non-pathogenic microorganism in low concentration. The medium is non-toxic, highly safe and advantageously used in high-risk patients such as elderly, hospitalized and immuno-compromised, AIDS patients without any side effects. The medium does not produce any side effects in babies or

pregnant women. The medium has abroad spectrum of therapeutic potential including reduction of DNA damage of the host cells.

Dwg.0/0

TECH

UPTX: 20040128

TECHNOLOGY FOCUS - FOOD - Preferred Components: The solid food ingredient a plant selected from vegetables, herbs, grains and fruits. The biocompatible liquid ingredient is selected from water, juice, milk and whey. The high protein ingredient is an offal product or a sea product. The vegetable ingredient is a fruit, a berry, a high protein, an herb, a beekeeping, a mash, and proteolytic ferment ingredients. The liquid ingredient (in %, by weight) (25-80), vegetable ingredient (1-30), fruit ingredient (1-20), berry ingredient (1-20), herb ingredient (1-15), high protein ingredient (1-30), beekeeping ingredient (0.1-5), sprouting grain ingredient (1-10), mash ingredient (1-15), and proteolytic ferment ingredient (0.1-1.0) are mixed. Sugar is added and dissolved in water. Then, a chemical ingredient selected from potassium, sodium, magnesium, and trace of cobalt, manganese, calcium and alcohol is added. The grain ingredient is obtained by sprouting the grains selected from rye, lentil, wheat, barley and beans, for 2-6 days at 20-30 degreesC in humid air environment. The sprouting grain ingredient is seeded with food grade fungi. The non-pathogenic microorganism is a bacteria or yeast. The non-pathogenic bacteria is selected from Lactobacilli, Bifidobacteria, Streptococci, Pedicocci, Leuconostoc, Propionic and acetic bacteria. The Lactobacilli bacteria is selected from *L. acidophilus*, *L. bifidus*, *L. brevis*, *L. bulgaricus*, *L. delbrucki*, *L. casei*, *L. cellobiosus*, *L. fermentum*, *L. gasserii*, *L. germentum*, *L. plantarum*, *L. reuteri*, *L. rhamnosus*, *L. sake*, *L. salivarioes*, *L. thermophilus* and *L. xylosus*. The Bifidobacteria is selected from *B. adolescentis*, *B. bifidum*, *B. breve*, *B. cereus*, *B. infantis*, *B. lactis*, *B. longum* and *B. thermophilus*. The vegetables ingredient is a leaf or root vegetables selected from cabbage, beet, rutabaga, carrot, pumpkin, spinach, beet, watermelon, melon, peanut, artichoke, eggplant, pepper sweet, asparagus, and tomato. The fruit ingredient is selected from apple, pear, kiwi, plum, citrus, grape, raisin, mango, guava, biwa, cornel, fig, cherry plum, quince, peach, pomegranate, avocado, pineapple, data, papaya, and banana. The berry ingredient is selected from raspberry, bilberry, guelder rose, dog rose, red ash berry, black ash berry, red currant, black currant, white currant, sea-buck-thorn berry, gooseberry, schizandra, blackberry, cowberry, bird cherry, cranberry, sweet cherry, cherry and straw berry. The herb ingredient is selected from ginseng, celery, parsley, dill, dandelion, nettle, and spinach. The offal ingredient is selected from spleen, kidney, heart, liver, brains, maw, and stomach. The beekeeping ingredient is selected from propolis, honey royal jelly, and **pollen** of flower. The proteolytic ferment ingredient is pepsin. The sugar ingredient is selected from glucose, fructose, sucrose, **mannose**, maltose, galactose, raffinose, corn syrup and lactose.

L13 ANSWER 7 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

AN 2003-370635 [35] WPIDS

CR 2002-499092 [53]

DNN N2003-295589 DNC C2003-098071

TI Lateral flow immunoassay device for detecting immune reactants, comprises a test strip having a sample site, colorimetric labeling site, and reaction sites each containing different allergens immobilized by a solubilizing agent.

DC B04 D16 S03

IN FORD, G M; HUBSCHER, T T; RUPPENTHAL, T M

PA (DEXA-N) DEXALL BIOMEDICAL LABS INC

CYC 1

PI US 6528325 B1 20030304 (200335)\* 9

ADT US 6528325 B1 US 2000-689682 20001013

PRAI US 2000-689682 20001013

AB US 6528325 B UPAB: 20030603

NOVELTY - A lateral flow immunoassay device, comprising a test strip which comprises:

- (a) a sample site for applying antibodies;
- (b) a colorimetric labeling site; and
- (c) reaction sites, each containing a different allergen such that when IgE antibodies labeled with colorimetric labeled anti-IgE antibodies come in contact with an antigen to which the IgE antibodies react, the reaction sites develop a colored line, indicating a positive response, is new.

DETAILED DESCRIPTION - A lateral flow immunoassay device for detecting immune reactants, comprising a test strip (7) which comprises:

- (a) a sample site for applying a sample comprising antibodies;
- (b) a colorimetric labeling site positioned downstream from the sample site, comprising colorimetric labeled anti-IgE antibody; and
- (c) reaction sites downstream from the labeling site, each containing a different allergen such that when IgE antibodies labeled with colorimetric labeled anti-IgE antibodies come in contact with an antigen to which the IgE antibodies react, the reaction sites develop a colored line, indicating a positive response, and where the allergens are immobilized to the test strip using solubilizing agent(s) present in an amount such that the allergen protein tertiary structure unfolds to allow for greater binding of the antigen to the test strip, and at least one of the solubilizing agents is a sugar or alcohol, is new.

USE - The invention is used for detecting immune reactants by applying a sample containing antibodies on the immunoassay device, and reading the immunoassay device (claimed). It is used for detecting the presence of antibodies in human or animal bodily fluids (e.g., blood, serum, plasma, **urine**, colostrum, milk, tears or saliva) to analytes such as bacteria, Chlamydiae, Rickettsiae, protozoa, allergens, autoimmune antigens, viral proteins, and carbohydrates.

ADVANTAGE - The invention provides a fast and effective lateral flow assay test for the testing of multiple-class specific antibodies. It can distinguish between reactive antibody contained in the classes of human antibody.

DESCRIPTION OF DRAWING(S) - The figure is a perspective view of a lateral flow immunoassay.

Test strip 7

Dwg.1/6

TECH

UPTX: 20030603

TECHNOLOGY FOCUS - INSTRUMENTATION AND TESTING - Preferred Device: The allergens include **pollens**, dust mite allergens, molds, animal epithelium, foods, and/or allergen mixes. At least one solubilizing agent is selected from sucrose, **mannose**, fructose, ethylene glycol, ethanol, methanol, glycerin, or dextran.

L13 ANSWER 8 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

AN 2002-527335 [56] WPIDS

DNC C2002-149274

TI Immunizing, modulating immune response, vaccinating against a disease, and sterilizing, a subject, by administering a composition of antigen and carbohydrate polymer comprising **mannose** to mucosal site of subject.

DC B04 D16

IN CHEERS, C; MCKENZIE, I F C; PIETERSZ, G A; STAMBAS, J; MCKENZIE, L F C

PA (AUST-N) AUSTIN RES INST; (UYME) UNIV MELBOURNE; (CHEE-I) CHEERS C;

(MCKE-I) MCKENZIE L F C; (PIET-I) PIETERSZ G A; (STAM-I) STAMBAS J

CYC 97

PI WO 2001093912 A1 20011213 (200256)\* EN 70  
 RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
 NL OA PT SD SE SL SZ TR TZ UG ZW  
 W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK  
 DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR  
 KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU  
 SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
 AU 2001063653 A 20011217 (200256)  
 EP 1301208 A1 20030416 (200328) EN  
 R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
 RO SE SI TR  
 US 2004043032 A1 20040304 (200417)  
 ADT WO 2001093912 A1 WO 2001-AU669 20010606; AU 2001063653 A AU 2001-63653  
 20010606; EP 1301208 A1 EP 2001-937864 20010606, WO 2001-AU669 20010606;  
 US 2004043032 A1 WO 2001-AU669 20010606, US 2003-297256 20030512  
 FDT AU 2001063653 A Based on WO 2001093912; EP 1301208 A1 Based on WO  
 2001093912  
 PRAI AU 2000-7977 20000606  
 AB WO 200193912 A UPAB: 20020903  
 NOVELTY - Immunizing (M1) a subject, modulating an immune response at a  
 mucosal site in a subject, vaccinating a subject against a disease and  
 sterilizing a non-human subject, involves administering a composition (I)  
 comprising an antigen (A) and a carbohydrate polymer (P) comprising  
**mannose** to a mucosal site of the subject.  
 DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for a  
 composition (I) adapted to be administered at a mucosal site of a subject,  
 to generate an immune response, comprising (A) and (P).  
 ACTIVITY - Ophthalmological; antibacterial; virucide;  
 tuberculostatic; fungicide; protozoacide.  
 MECHANISM OF ACTION - Inducer of Th1/Th2 cytokine response (claimed);  
 vaccine.  
 (CBA x BALB/c) F1 mice were immunized intranasally with M-LLOP  
 (mannan-listeriolysin O (LLO) glutathione-S-transferase (GST) fusion  
 protein (LLOP) conjugate), and tears and lung washings were collected on  
 days 24 and 35, respectively. Mice immunized with M-LLOP conjugate  
 produced significantly higher titres of IgA (p less than 0.01) in tears  
 when compared with the unconjugated controls. The same trend was observed  
 in the lungs at day 35. Significantly higher titers of IgA were detected  
 in mice immunized with M-LLOP (1/975) compared with LLOP alone (1/38) (p  
 less than 0.05).  
 USE - The method is useful for immunizing a subject, modulating an  
 immune response at a mucosal site of a subject, vaccinating a subject and  
 sterilizing a non-human subject. (I) is useful in the manufacture of a  
 medicament for the modulation of an immune response in a subject and in  
 the manufacture of a vaccine suitable for administration to a mucosal site  
 (claimed).  
 (I) is useful for treating diseases or **infection** of the  
 eyes such as trachoma or conjunctivitis, listeriosis, tuberculosis,  
 influenza, colds, respiratory diseases, sexually transmitted diseases or  
 infetions by viruses, bacteria, fungi, protozoa or other microorganisms or  
 pathogens.  
 Dwg. 0/15  
 TECH UPTX: 20020903  
 TECHNOLOGY FOCUS - PHARMACEUTICALS - Preferred Method: The immune response  
 further involves stimulation of mediators of cellular immunity such as Th1  
 and/or Th2. The mucosal or secretory immune response is stronger than a  
 systemic immune response.  
 Preferred Antigen: (A) is selected from **pollens**, allergens,  
 bacteria, viruses, yeast, fungi, protozoa, or other microorganisms  
 including pathogens of humans, animals and plants. Alternatively, (A) is

selected from any one of the 37 antigens given in the specification, e.g. influenza virus, haemagglutinin of influenza, Porphyromona gingivalis, proteinase and adhesin epitopes of Porphyromona gingivalis, Helicobacter pylori, urease of Helicobacter pylori, rotavirus, recombinant VP5 protein of rotavirus, HIV, gp120 of HIV, respiratory syncytial virus (RSV), surface proteins of RSV and ovalbumin peptides and Listeria monocytogenes, Mycobacterium tuberculosis, Mycobacterium avium, BCG, influenza nucleoprotein, RSV F or G proteins, Candida albicans and Chlamydia trachomatis or their outer membrane proteins, Neisseria meningitidis class 1 outer protein, Herpes simplex virus type I glycoprotein G or gp D or CP27, Human Papilloma Virus, Murray valley encephalitis virus E glycoprotein, or their antigenic portions and derivatives.

(A) is conjugated to oxidized mannan.

L13 ANSWER 9 OF 9 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 AN 1999-254927 [21] WPIDS  
 DNC C1999-074606  
 TI Immunoregulatory composition comprising **mannose** receptor-bearing cells, an antigen and **mannose**, useful for prevention/treatment of cancer.  
 DC B04 D16  
 IN APOSTOLOPOULOS, V; MCKENZIE, I F C; PIETERSZ, G A  
 PA (AUST-N) AUSTIN RES INST  
 CYC 82  
 PI WO 9916455 A1 19990408 (199921)\* EN 82  
 RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL  
 OA PT SD SE SZ UG ZW  
 W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE  
 GH GM HR HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG  
 MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG  
 UZ VN YU ZW  
 AU 9894555 A 19990423 (199935)  
 EP 1027063 A1 20000816 (200040) EN  
 R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI NL PT SE  
 JP 2001517709 W 20011009 (200174) 75  
 AU 754065 B 20021107 (200302)  
 ADT WO 9916455 A1 WO 1998-IB1718 19980929; AU 9894555 A AU 1998-94555  
 19980929; EP 1027063 A1 EP 1998-947738 19980929; WO 1998-IB1718 19980929;  
 JP 2001517709 W WO 1998-IB1718 19980929; JP 2000-513589 19980929; AU  
 754065 B AU 1998-94555 19980929  
 FDT AU 9894555 A Based on WO 9916455; EP 1027063 A1 Based on WO 9916455; JP  
 2001517709 W Based on WO 9916455; AU 754065 B Previous Publ. AU 9894555,  
 Based on WO 9916455  
 PRAI US 1997-60594P 19970929  
 AB WO 9916455 A UPAB: 20011203  
 NOVELTY - An immunoregulatory composition (I) comprising isolated **mannose** receptor-bearing cells and a conjugate comprising an antigen and a **mannose** selected from fully oxidized and partially reduced **mannose** having aldehydes.  
 DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:  
 (1) a composition (II) comprising an immunoregulatory **mannose** -receptor bearing cell population, derived by culturing **mannose** -receptor bearing cells under conditions comprising an antigen delivery system;  
 (2) an immunoregulatory **mannose** receptor-bearing cell population, derived by culturing **mannose** receptor-bearing cells in vitro with biological response modifiers to enhance the population, then incubating with (I);  
 (3) a mucin antigen delivery vehicle, comprising (I);

(4) a method (II) for obtaining a population comprising immunoregulatory **mannose** receptor-bearing cells, comprising culturing with an antigen delivery medium; and

(5) a method (III) for inducing an immune response comprising administering (I).

USE - (I) is useful as a therapeutic agent in animals with natural antibodies against mucin, for inducing a cell mediated immune response specifically to mucin. It is useful for prevention/treatment of tumors, particularly adenocarcinoma, more particularly breast cancer.

ADVANTAGE - Prior art methods for removal of tumors uses disfiguring and costly surgery, and/or chemotherapeutic and radiation methods with severe side-effects. The new composition is non-toxic  
Dwg.0/14

TECH

UPTX: 19990603

TECHNOLOGY FOCUS - BIOLOGY - Preferred Composition: (I) preferably has a partially reduced **mannose** having aldehydes, and the receptor-bearing cells are derived from cells selected from peripheral blood leukocytes, bone marrow, stem cells, tumor cells, stromal cells, peritoneal cells, spleen, lung and lymph node cells. The cells comprise cells enriched for macrophage or dendritic cells. The cells comprise cells that express molecules selected from **mannose** receptor, CD11b, CD14, CD68, CD80 and CD86. The cells are combined with the conjugate in vitro or in vivo. The cells comprise cells that have been contacted with at least one biological response modifier, which are capable of inducing **mannose** receptors on capable cells. The modifier is a cytokine or vitamin, selected from GM-CSF, interleukin-3, interleukin-4, vitamin D, GM-CSF, Flt-3 ligand and TNFalpha.

The **mannose** is a conformational or configurational isomer of **mannose**, and comprises a carbohydrate polymer of at least 2 units. The antigen delivery medium of (II) comprises a conjugate comprising an antigen and **mannose** selected from fully oxidized and preferably partially reduced **mannose** having aldehydes. The **mannose** comprises a carbohydrate polymer comprised of at least 2 carbohydrate units, where the cells have been incubated with at least one biological response modulators prior to culturing, preferably GM-CSF, interleukin-3, interleukin-4, vitamin D, GM-CSF, Flt-3 ligand and TNF alpha, and culturing in performed in vitro.

Preferred Antigen: The antigen is selected from nm23, p53, Her2.neu, MUC1, BRACA1 and 2, MAGE antigen, CEA, Erb2, **pollen**, hepatitis C virus (HIV) core, E1, E2 and NS2 proteins, Plasmodium falciparum circumsporozoite protein, HIV-gp120/160 envelope glycoprotein, Streptococcus surface protein Ag, influenza nucleoprotein, hemagglutinin-neuraminidase surface **infection**, TcpA pilin subunit, VP1 protein, LMCV nucleoprotein, Leishmania major surface glycoprotein (gp63), Bordetella pertussis surface protein, rabies virus G protein, Streptococcus M protein, respiratory syncytial virus (RSV) F or G proteins, Epstein Barr virus (EBV) gp340 or nucleocapsid protein 3A, hemagglutinin, Borrelia burgdorferi outer surface protein (Osp) A, Mycobacterium tuberculosis 38 kDa lipoprotein or Ag85, Neisseria meningitidis class 1 outer protein, Varicella zoster virus IE62 and gp1, Rubella virus capsid protein, Hepatitis B virus pre S1 ag, Herpes simplex virus type I glycoprotein G or gpD or CP27, Murray valley encephalitis virus E glycoprotein Hepatitis A virus VP1, polio virus capsid protein VP1, -2, and -3, Chlamydia trachomatis surface protein, Hepatitis B virus envelope Ag pre S2, Human rhinovirus (HRV) capsid, papillomavirus peptides from oncogene E6 and E7, Listeria surface protein, Varicella virus envelope protein, Vaccinia virus envelope protein, Brucella surface protein, a combination of at least one of the above antigens, an amino acid subunit of the antigens comprising at least 5 amino acids, or a combination of subunits, or the antigen is a mucin polypeptide, or at

least one repeated unit or a fragment of. The antigen is preferably human mucin, comprising 2-80 repeated subunits. The subunits may comprise part of a fusion polypeptide.

Preferred Population: The **mannose** receptor-bearing cell population is cultured in vitro using method (II) for 1-6 (preferably 3) hours, and incubated for 10-30 (preferably 16-24) hours. The cells are enriched for macrophage cells or dendritic cells, and the biological response modifier (GM-CSF, interleukin-3, interleukin-4, vitamin D, GM-CSF, Flt-3 ligand and TNF alpha) increases the number of **mannose** receptors on the cells. The **mannose** is partially reduced **mannose** having aldehydes, and the antigen comprises (human) mucin.

Preferred Method: (III) induces a cell mediated immune response, using **mannose** receptor-bearing cells with an MHC matched to the recipient animal, the donor being related/unrelated.

L20 ANSWER 1 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2004-710398 [70] WPIDS  
 DOC. NO. CPI: C2004-250569  
 TITLE: Vaccine composition useful for treating or preventing group A Streptococcal **infection** in individual, comprises one or more of group A Streptococcal antigen and proteosome adjuvant.  
 DERWENT CLASS: B04 D16  
 INVENTOR(S): BATZLOFF, M R; BURT, D S; GOOD, M F; LEANDERSON, T B; LOWELL, G H; **WHITE, G L**  
 PATENT ASSIGNEE(S): (BURT-I) BURT D; (COUN-N) COUNCIL QUEENSLAND INST MEDICAL RES; (LEAN-I) LEANDERSON T; (LOWE-I) LOWELL G; (WHIT-I) **WHITE G**; (IDBI-N) ID BIOMEDICAL CORP QUEBEC  
 COUNTRY COUNT: 2  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
AU 2002302132	A1	20040603	(200470)*		63
US 2005002956	A1	20050106	(200504)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
AU 2002302132	A1	AU 2002-302132	20021115
US 2005002956	A1 Provisional	US 2002-426409P	20021115
		US 2003-706275	20031113

PRIORITY APPLN. INFO: AU 2002-302132 20021115

AB AU2002302132 A UPAB: 20041101

NOVELTY - A vaccine composition (I) comprises one or more of group A Streptococcal antigen and a proteosome adjuvant.

ACTIVITY - Antibacterial.

MECHANISM OF ACTION - Vaccine (claimed). Immunogenicity of the J14 peptide/proteosome adjuvant was determined in the murine model. The potential of intranasal immunization with the vaccine candidate and degree of protection inferred against intraperitoneal challenge were determined. Peptides complexed with proteosome adjuvant were used for intranasal immunization of Swiss outbred mice. Two boosts of 60 micro g peptide/30

micro l/mouse at 21 day intervals, followed the primary immunization. One day prior to the boosts and 15 days after serum IgG titres specific for J14 were measured and isotyping performed. IgA titres from salivations collected 2 days prior and 14 days after boosts were determined. Mice were intraperitoneally challenged with a dose of M1 group A streptococcal (GAS) reference strain. Peptide-proteosome adjuvant group induced significant J14 specific serum antibody titres. Overall, the average titres of the control groups were not significant compared to the J14-adjuvant groups in the final bleed. Approximately, one third of group 4 (mice immunized with the carboxyl terminal anchored J14) induced antibodies that recognized the J14 peptide.

USE - (I) is useful for the treatment or prophylaxis of a group A Streptococcal **infection** in an individual which involves administering (I) to the individual intranasally, where the treatment is effected through reduction or prevention of bacterial colonization of the throat (claimed).

Dwg.0/28

L20 ANSWER 2 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2004-570523 [55] WPIDS  
 DOC. NO. CPI: C2004-208289  
 TITLE: Use of D-mannose for maintenance of  
 urinary tract health in the face of  
 infection e.g. E. Coli **infection**.  
 B03 B04  
 DERWENT CLASS:  
 INVENTOR(S): ONEAL, J; WHITE, G  
 PATENT ASSIGNEE(S): (ONEA-I) ONEAL J; (WHIT-I) WHITE G  
 COUNTRY COUNT: 1  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
US 2004147459	A1	20040729	(200455)*		6

#### APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
US 2004147459	A1 Provisional	US 2002-420696P	20021023
		US 2003-691423	20031022

PRIORITY APPLN. INFO: US 2002-420696P 20021023; US  
 2003-691423 20031022

AB US2004147459 A UPAB: 20040826

NOVELTY - Maintenance of **urinary** tract health in the face of **infection** involves administering a dosage of 1 - 2 teaspoons of D-mannose three times a day with meals for 1 - 2 weeks or until the symptoms subside.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is included for a composition comprising a therapeutic dosage of D-mannose and a therapeutic dosage of at least one of an extract of **Crataeva nurvala**, **willow bark** or pollen extract simultaneously with D-mannose.

ACTIVITY - Uropathic; Antimicrobial.

MECHANISM OF ACTION - E. coli urethral epithelial cell attachment inhibitor.

USE - For maintaining **urinary** tract health in the face of **infection**; in combination with a capsule containing herbs that affect the **urinary** tract; and for dealing with a **urinary**



tract infection (all claimed). The urinary tract infection include E. coli infection.

ADVANTAGE - The use of D-mannose in the maintenance of urinary tract health in the face of infection and in the treatment of urinary tract infection preferentially provides attachment of E. coli fimbriae to the administered D-mannose present in the urine, rather than attachment to D-mannose in the epithelial cells of the urinary tract. This results in the E. coli bacteria surrounded by the molecules of D-mannose and promotes their natural elimination by mechanical and not pharmacological action. The few remaining bacteria can then be better handled by the body's natural defenses, the white blood cells. Mannose can not be broken down in the body, and thus is safe for diabetics, pregnant women and the elderly, and is virtually free from the risk of overdose. The method does not involve the use of antibiotics and hence avoids the side effects and resistant strain development associated with them. The administration regimen provides a quantity of mannose sufficient to remove a majority of E/ coli in the urinary tract, while improving the ease of use and compliance. The dosages are effective in doctor-run trials.

Dwg.0/3

L20 ANSWER 3 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2003-671302 [63] WPIDS  
 DOC. NO. NON-CPI: N2003-536060  
 DOC. NO. CPI: C2003-183016  
 TITLE: Intraluminal stent or graft useful for treating atherosclerosis comprises a tubular body including several unit cells, extends from proximal to distal end, and is capable of expanding from a radially compressed state to expanded state.  
 DERWENT CLASS: A96 B05 D22 P32  
 INVENTOR(S): WHITE, G H  
 PATENT ASSIGNEE(S): (WHIT-I) WHITE G H  
 COUNTRY COUNT: 102  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003053284	A1	20030703	(200363)*	EN	23
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2002351883	A1	20030709	(200428)		
US 2005182477	A1	20050818	(200555)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003053284	A1	WO 2002-AU1757	20021220
AU 2002351883	A1	AU 2002-351883	20021220
US 2005182477	A1	WO 2002-AU1757	20021220
		US 2005-499016	20050425

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002351883	A1 Based on	WO 2003053284

PRIORITY APPLN. INFO: AU 2001-9694 20011220; AU  
2001-9693 20011220

AB WO2003053284 A UPAB: 20031001

NOVELTY - An intraluminal stent (S1) or graft (G1) comprises a tubular body extending from proximal to distal end, is capable of expanding or being expanded from a radially compressed state to a radially expanded state, and includes several unit cells. Each unit cell has a first and a second end portion, with greater dimension of the first end portion, and each optionally comprising tapering regions and a longitudinal and a traverse axis.

DETAILED DESCRIPTION - An intraluminal stent (S1) comprises a tubular body extending from proximal to distal end, and is capable of expanding or being expanded from a radially compressed state to a radially expanded state. (S1) comprises several unit cells and (G1) is circumferentially reinforced along at least a part of its length by several unit cells. Each unit cell has either a first end portion and a second end portion, where the first end portion has greater dimensions than the second end portion, and a longitudinal axis and a traverse axis, where each unit cell is symmetrical about its longitudinal axis and asymmetrical about the traverse axis; or each unit cell comprises a first end portion comprising several tapering regions and a second end portion comprising at least one tapering region.

An INDEPENDENT CLAIM is also included for a method of positioning (S1) or (G1) in a vessel of a patient involving:

- (a) introducing a catheter or other delivery device into a vein, artery or other vessel in the body of a patient when the tubular body of (S1) or (G1) is in its radially compressed state;
- (b) causing (S1) or (G1) to be carried through the catheter or other delivery device to a target site of stenosis in a vessel;
- (c) causing or allowing the tubular of the intraluminal stent to expand within the vessel; and
- (d) withdrawing the catheter or other delivery device along with any other apparatus used to introduce (S1) or (G1) into the vessel from the body of the patient.

USE - For treating stenosis or other conditions of the visceral arteries such as renal and mesenteric arteries, the iliac artery and the sub-clavian artery and stenotic lesions in the peripheral vasculature, the coronary circulation, the hepato-biliary and genito-urinary tracts; and aneurysmal disease of arteries of a patient including the aorta, renal and mesenteric arteries, the iliac artery, the sub-clavian artery and diseases of the peripheral vasculature and the coronary circulation (all claimed) such as atherosclerosis.

ADVANTAGE - (S1) And (G1) is effective in providing a stable bridge for the flow of blood through a disease section of the vessel, and has good strength and flexibility with good expansile ratio. The graft can be packaged in a compressed form into a suitable introducer catheter while at the same time provides an expanded form of suitable diameter to engage the wall of the vessel in which it is placed. The unit cells have curved or sinusoidal shape sop that any length change during radial compression of the stent or the graft is compensated for by the spring-like properties of the unit cells. The radially compressed state of the tubular body enables the delivery of the stent through an introducer catheter.

DESCRIPTION OF DRAWING(S) - The figure shows a side elevational view of the stent.

Intraluminal stent 10

Tubular body 11  
 Proximal end 12  
 Distal end 13  
 Unit cell 14  
     First end portion 15  
 First end 16  
     Second end portion 17  
 Second end 18  
     Tapering regions 21  
     Terminating points 22  
     Tapering region of the second end 23  
     Circumferential series 25a, 25b  
     Common sides 28a, 28b  
 Dwg.1/15

L20 ANSWER 4 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2003-607877 [57] WPIDS  
 DOC. NO. NON-CPI: N2003-484708  
 DOC. NO. CPI: C2003-165605  
 TITLE: Engagement device for intraluminal graft or stent has  
     main body which expands from radially compressed state to  
     expanded state such that, in expanded state, at least  
     part of the body engages wall of vessel in which the  
     device is positioned.  
 DERWENT CLASS: A96 B05 D22 P32  
 INVENTOR(S): WHITE, G H  
 PATENT ASSIGNEE(S): (WHIT-I) WHITE G H  
 COUNTRY COUNT: 103  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003053283	A1	20030703	(200357)*	EN	31
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU					
MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK					
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR					
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT					
RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA					
ZM ZW					
AU 2002350277	A1	20030709	(200428)		
EP 1465550	A1	20041013	(200467)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC					
MK NL PT RO SE SI SK TR					
US 2005171599	A1	20050804	(200552)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003053283	A1	WO 2002-AU1725	20021219
AU 2002350277	A1	AU 2002-350277	20021219
EP 1465550	A1	EP 2002-784919	20021219
		WO 2002-AU1725	20021219
US 2005171599	A1	WO 2002-AU1725	20021219
		US 2005-499017	20050401

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
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AU 2002350277      A1 Based on              WO 2003053283  
EP 1465550          A1 Based on              WO 2003053283

PRIORITY APPLN. INFO: AU 2001-9692              20011220

AB WO2003053283 A UPAB: 20030906

NOVELTY - Engagement device for an intraluminal graft or stent has a main body (11) which expands from a radially compressed state to a radially expanded state such that, when in its expanded state, at least part of the main body engages a wall of a vessel in which the engagement device is positioned. The main body has a receiving section (12) to receive and engage at least a portion of the graft or stent.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an intraluminal assembly for placement in a vessel (14) of a patient, which comprises an engagement device, and an intraluminal graft or stent; and

(2) a method of positioning an engagement device for an intraluminal graft or stent in a diseased vessel of a patient, which includes introducing a catheter (24) or other delivery device into a vein, artery, or other vessel of a patient until a distal end of the catheter or other delivery device is positioned at or adjacent a target region of the diseased vessel; causing the engagement device for an intraluminal graft or stent to be carried through the catheter or other delivery device to the target region, with the main body of the device in its radially compressed state; releasing the main body from the distal end of the catheter or other delivery device and causing or allowing the main body to move to its radially expanded state within the target region of the diseased vessel such that at least part of the main body engages a wall of the target region of the vessel; positioning a radially compressed intraluminal graft or stent at least partially within the receiving region of the main body; causing or allowing the intraluminal graft or stent to move to a radially expanded state such that at least a portion of the intraluminal stent or graft engages with the receiving region of the main body; and withdrawing the catheter or other delivery device along with any other apparatus used to introduce the engagement device for an intraluminal graft or stent and/or the intraluminal graft or stent from the patient.

USE - The engagement device is used for securing or sealing an intraluminal graft or stent in place within a vessel of a patient. The intraluminal graft or stent is used in the grafting or stenting of diseased arteries of a patient including the aorta and the visceral arteries e.g., the renal and mesenteric arteries, the iliac artery, and the sub-clavian artery; or of the peripheral vasculature, the coronary circulation, the hepato-biliary, and genito-urinary tracts (claimed).

ADVANTAGE - The inventive device eliminates the problems associated with conventional intraluminal grafts which do not align or seal well with a diseased or anatomically unique vessel structure and which have the tendency to slip within the vessel, causing the graft to occlude an opening of a pre- or post-branching vessel.

DESCRIPTION OF DRAWING(S) - The figure depicts delivery of the inventive device to a target vessel.

Main body 11

Receiving section 12

Vessel 14

Outer wall 16

Internal wall 17

Catheter 24

Dwg.2b/9

L20 ANSWER 5 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2002-227039 [28] WPIDS  
 DOC. NO. CPI: C2002-069098  
 TITLE: Treating e.g. neuropathic pain, comprises administration  
 of new and known compounds which are high potency  
 capsaicin receptor antagonists, and are not capsaicin  
 analogs.  
 DERWENT CLASS: B02 B03 B07  
 INVENTOR(S): BAKTHAVATCHALAM, R; DESIMONE, R W; HODGETTS, K J;  
 HUTCHINSON, A; KRAUSE, J E; WHITE, G G;  
 HUTCHISON, A  
 PATENT ASSIGNEE(S): (DESI-I) DESIMONE R W; (HODG-I) HODGETTS K J; (HUTC-I)  
 HUTCHISON A; (KRAU-I) KRAUSE J E; (NEUR-N) NEUROGEN CORP;  
 (WHIT-I) WHITE G G; (BAKT-I) BAKTHAVATCHALAM R  
 COUNTRY COUNT: 97  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002008221	A2	20020131	(200228)*	EN	209
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001080667	A	20020205	(200236)		
US 2002132853	A1	20020919	(200264)		
EP 1301484	A2	20030416	(200328)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
KR 2003024799	A	20030326	(200346)		
BR 2001012631	A	20030923	(200373)		
CN 1443170	A	20030917	(200382)		
US 6723730	B2	20040420	(200427)		
JP 2004525071	W	20040819	(200455)	337	
US 2004176443	A1	20040909	(200459)		
NZ 523526	A	20041029	(200474)		
MX 2003000458	A1	20040601	(200504)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002008221	A2	WO 2001-US22930	20010720
AU 2001080667	A	AU 2001-80667	20010720
US 2002132853	A1 Provisional	US 2000-219529P	20000720
	Provisional	US 2000-230726P	20000907
	Provisional	US 2001-280223P	20010330
		US 2001-910442	20010720
EP 1301484	A2	EP 2001-959075	20010720
		WO 2001-US22930	20010720
KR 2003024799	A	KR 2003-700866	20030120
BR 2001012631	A	BR 2001-12631	20010720
		WO 2001-US22930	20010720
CN 1443170	A	CN 2001-813046	20010720
US 6723730	B2 Provisional	US 2000-219529P	20000720
	Provisional	US 2000-230726P	20000907
	Provisional	US 2001-280223P	20010330
		US 2001-910442	20010720

JP 2004525071	W	WO 2001-US22930	20010720
		JP 2002-514127	20010720
US 2004176443	A1 Provisional	US 2000-219529P	20000720
	Provisional	US 2000-230726P	20000907
	Provisional	US 2001-280223P	20010330
	Cont of	US 2001-910442	20010720
		US 2004-799286	20040312
NZ 523526	A	NZ 2001-523526	20010720
		WO 2001-US22930	20010720
MX 2003000458	A1	WO 2001-US22930	20010720
		MX 2003-458	20030116

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001080667	A Based on	WO 2002008221
EP 1301484	A2 Based on	WO 2002008221
BR 2001012631	A Based on	WO 2002008221
JP 2004525071	W Based on	WO 2002008221
US 2004176443	A1 Cont of	US 6723730
NZ 523526	A Based on	WO 2002008221
MX 2003000458	A1 Based on	WO 2002008221

PRIORITY APPLN. INFO: US 2001-280223P 20010330; US  
 2000-219529P 20000720; US  
 2000-230726P 20000907; US  
 2001-910442 20010720; US  
 2004-799286 20040312

AB WO 200208221 A UPAB: 20021031

NOVELTY - Treating at least one symptom selected from e.g.

burns or irritation due to exposure to heat or light, bronchoconstriction or irritation due to exposure to tear gas, and neuropathic pain and peripheral nerve mediated pain comprises administering a compound that is a high potency capsaicin receptor antagonist in an in vitro assay of capsaicin receptor antagonism, and is not a capsaicin analog.

DETAILED DESCRIPTION - Treating at least one symptom selected from symptoms of exposure to capsaicin, burns or irritation due to exposure to heat or light, burns, bronchoconstriction or irritation due to exposure to tear gas, burns or irritation due to exposure to acid and treating neuropathic pain and peripheral nerve mediated pain comprises administering a dose of a compound that is a high potency capsaicin receptor antagonist in an in vitro assay of capsaicin receptor antagonism, and is not a capsaicin analog.

INDEPENDENT CLAIMS are included for compounds of formula (I)-(IV) and their salts.

A = e.g. absent or is O, S, NRA, CRBRB', NRACRBRB' or CRBRB'NRA;

RA, RB, RB' = H or alkyl;

Z = O or S;

R1, R2 = H or alkyl;

R3, R4 = e.g. alkyl, alkenyl, alkynyl, alkoxy, mono or dialkylamino, alkylthio or alkyl ketone (all optionally substituted);

Ar1, Ar2 = e.g. cycloalkyl, 5-8 membered heterocyclylalkyl (containing 1-3 heteroatoms of N, O or S) or aryl having 1-3 rings (all optionally substituted);

Ar1 asterisk, Ar2 asterisk = e.g. cyclohexyl, cyclopentyl, piperidinyl, piperazinyl, phenyl or benzo(b)thiophenyl, (all optionally substituted);

x = 1 or 3;

Ar1'', Ar2'' = e.g. cyclohexyl, cyclopentyl, piperidinyl, piperazinyl, pyrrolyl or quinolinyl, (where Ar1'' and Ar2'' are both optionally substituted);

G, Q, T, W = e.g. N or CH;

R3 asterisk, R4 asterisk = e.g. 1-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, 1-6C alkoxy, mono or di(1-6C)alkylamino or aryl having 1-3 rings, (all optionally substituted);

R5 asterisk = e.g. 3-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, 3-6C alkoxy or NH(1-6C alkyl) (all optionally substituted); and

R9 asterisk = e.g. 1-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, 1-6C alkoxy or NH(1-6C alkyl) (all optionally substituted) or halo.

The full definitions are given in the DEFINITIONS (Full Definitions) Field.

ACTIVITY - Analgesic; Vulnerary; Uropathic; Antipsoriatic; Dermatological; Antiallergic.

In an assay to assess the effectiveness of the compounds in relieving one of the symptoms of neuropathic pain produced by unilateral mononeuropathy (namely mechanical hyperalgesia), the compounds produced a reduction in mechanical hyperalgesia elicited by a pin-prick stimulus in rats with a chronic constriction injury, at doses of 50 mg/kg or less.

MECHANISM OF ACTION - Capsaicin receptor modulator; Capsaicin receptor antagonist; Vanilloid binding inhibitor.

USE - For treating at least one symptom selected from symptoms of exposure to capsaicin, burns or irritation due to exposure to heat or light, burns, bronchoconstriction or irritation due to exposure to tear gas, burns or irritation due to exposure to acid and treating neuropathic pain and peripheral nerve mediated pain (e.g. pain associated with post-mastectomy pain syndrome, stump pain, phantom limb pain, oral neuropathic pain, Charcot's pain, toothache, venomous snake bite, spider bite, insect sting, postherpetic neuralgia, diabetic neuropathy, reflex sympathetic dystrophy, trigeminal neuralgia, osteoarthritis, rheumatoid arthritis, fibromyalgia, Guillain-Barre syndrome, meralgia paresthetica, burning mouth syndrome, bilateral peripheral neuropathy, causalgia, sciatic neuritis, peripheral neuritis, polyneuritis, optic neuritis, postfebrile neuritis, migrating neuritis, segmental neuritis, Gombault's neuritis, neuronitis, cervicobrachial neuralgia, cranial neuralgia, geniculate neuralgia, glossopharyngeal neuralgia, migranous neuralgia, idiopathic neuralgia, intercostal neuralgia, mammary neuralgia, mandibular joint neuralgia, Morton's neuralgia, nasociliary neuralgia, occipital neuralgia, red neuralgia, Sluder's neuralgia, splenopalatine neuralgia, supraorbital neuralgia, vidian neuralgia, sinus headache, tension headache, labor, childbirth, intestinal gas, menstruation, cancer and trauma); and for reducing the calcium conductance of a capsaicin receptor, (all claimed). Also useful for as tools for the analysis of type I vanilloid receptors (VRI) and as probes for the quantitative measurement and localization of VRI receptors in cell and tissue samples. Also useful for reducing the frequency of urinary incontinence (e.g. caused by detrusor hyperflexia of spinal origin or bladder hypersensitivity) and for treating itching conditions (e.g. psoriatic pruritus, itch due to hemodialysis, aquagenic pruritus, itching associated with vulvar vestibulitis, contact dermatitis or skin allergies).

Dwg.0/0

L20 ANSWER 6 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2001-557626 [62] WPIDS

DOC. NO. CPI: C2001-165808

TITLE: New improved vaccines, which comprise proteosomes and protein antigens, useful against influenza, particularly for protecting humans against influenza infection

DERWENT CLASS: B04 D16  
 INVENTOR(S): BURT, D S; FRIES, L F; JONES, D H; LOWELL, G H; PLANTE, M; TOROSSIAN, K; **WHITE, G L**  
 PATENT ASSIGNEE(S): (INTE-N) INTELLIVAX INT INC; (IDBI-N) ID BIOMEDICAL CORP QUEBEC; (BURT-I) BURT D S; (FRIE-I) FRIES L F; (JONE-I) JONES D H; (LOWE-I) LOWELL G H; (PLAN-I) PLANTE M; (TORO-I) TOROSSIAN K; (WHIT-I) WHITE G L  
 COUNTRY COUNT: 95  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001060402	A2	20010823	(200162)*	EN	44
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001038478	A	20010827	(200176)		
US 2001053368	A1	20011220	(200206)		
NO 2002003829	A	20020813	(200277)		
EP 1255561	A2	20021113	(200282)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
JP 2003522802	W	20030729	(200358)		66
EP 1419784	A2	20040519	(200433)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE TR					
US 6743900	B2	20040601	(200436)		
US 2004156867	A1	20040812	(200454)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001060402	A2	WO 2001-US5222	20010215
AU 2001038478	A	AU 2001-38478	20010215
US 2001053368	A1 Provisional	US 2000-182476P	20000215
		US 2001-788280	20010215
NO 2002003829	A	WO 2001-US5222	20010215
		NO 2002-3829	20020813
EP 1255561	A2	EP 2001-910923	20010215
		WO 2001-US5222	20010215
JP 2003522802	W	JP 2001-559498	20010215
		WO 2001-US5222	20010215
EP 1419784	A2 Div ex	EP 2001-910923	20010215
		EP 2004-953	20010215
US 6743900	B2 Provisional	US 2000-182476P	20000215
		US 2001-788280	20010215
US 2004156867	A1 Provisional Div ex	US 2000-182476P	20000215
		US 2001-788280	20010215
		US 2004-771737	20040203

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001038478	A Based on	WO 2001060402
EP 1255561	A2 Based on	WO 2001060402
JP 2003522802	W Based on	WO 2001060402





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WO 2001030270  A2 20010503 (200134)* EN 21
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ
NL OA PT SD SE SL SZ TZ UG ZW
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE
SG SI SK SL TJ TM TR TT TZ UA UG
AU 2001037889  A 20010508 (200149)
EP 1214021     A2 20020619 (200240) EN
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT
RO SE SI
US 2003033002  A1 20030213 (200314)
AU 778349      B2 20041202 (200506)
US 6849088     B2 20050201 (200511)#
US 2006015176  A1 20060119 (200607)

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## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001030270	A2	WO 2000-US26239	20000925
AU 2001037889	A	AU 2001-37889	20000925
EP 1214021	A2	EP 2000-991996	20000925
		WO 2000-US26239	20000925
US 2003033002	A1 CIP of	US 1998-163580	19980930
	CIP of	US 1998-204699	19981203
		US 2001-55787	20011107
AU 778349	B2	AU 2001-37889	20000925
US 6849088	B2 CIP of	US 1998-163580	19980930
	CIP of	US 1998-204699	19981203
		US 2001-55787	20011107
US 2006015176	A1 Cont of	US 2000-595043	20000615
		US 2005-173322	20050630

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001037889	A Based on	WO 2001030270
EP 1214021	A2 Based on	WO 2001030270
US 2003033002	A1 CIP of	US 6368345
AU 778349	B2 Previous Publ.	AU 2001037889
	Based on	WO 2001030270
US 6849088	B2 CIP of	US 6368345

PRIORITY APPLN. INFO: AU 1999-3029 19990923

AB WO 200130270 A UPAB: 20060130

NOVELTY - An intraluminal graft (10) comprises of a tubular body (22) of a predetermined, non linear shape. The graft can be introduced into e.g. an aortic aneurysm (11) in the aorta (12) from a catheter through one of the femoral arteries (16) in the aorta. The tubular body shape allows the graft to conform to the natural or pathological contours of the aorta.

DETAILED DESCRIPTION - The tubular body is preferably formed from woven Dacron, interwoven with spaced apart reinforcing wires. An INDEPENDENT CLAIM is also included for a method for emplacing an intraluminal device.

USE - For treating aneurysms of e.g. femoral artery, popliteal artery, thoracic segment of aorta, or visceral arteries e.g. renal and mesenteric arteries, iliac artery, sub clavian artery. Also for treating

stenotic lesions in peripheral vasculature.

ADVANTAGE - Graft can be used for treating stenotic lesions in other vessels e.g. hepato biliary and genito **urinary** tracts, or vessels which constitute coronary circulation. Graft can conform or securely fit into wall of vessel e.g. aorta, without possible dislodging, when aneurysm in aorta expands. Graft can also align with both non linear and substantially linear vessels. Allows vessel with graft to be imaged by e.g. ultrasound, plain abdominal film, computerized tomography CT scanning. Enhances patient's vasculature.

DESCRIPTION OF DRAWING(S) - The figure shows the partially cut away ventral view of the patient, with the intraluminal graft bridging the aortic aneurysm of the patient. Another figure shows the alternate embodiment of the intraluminal graft.

Intraluminal graft 10  
Aortic aneurysm 11  
Aorta 12  
Femoral arteries 16  
Tubular body 22  
Dwg.1,9/9

L20 ANSWER 8 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
ACCESSION NUMBER: 1994-168911 [21] WPIDS  
CROSS REFERENCE: 1986-253146 [39]; 1987-095565 [14]; 1994-137461 [17]  
DOC. NO. CPI: C1994-077302  
TITLE: New therapeutic combination of 3'-azido-3'-deoxy  
thymidine and a second agent - useful in the treatment of  
human retroviral **infection** especially HTLV-1, HTLV-2  
and HIV **infections**..  
DERWENT CLASS: B03 B05  
INVENTOR(S): BARRY, D W; CLEMONS, R H; DE MIRANDA, P M; FREEMAN, G A;  
FURMAN, P A; KING, D H; KIRK, L E; LEHRMAN, S N; RIDEOUT,  
J L; SHAVER, S R; ST CLAIR, M H; **WHITE, G**;  
WOLBERG, G C; ZIMMERMAN, T P; WOLBERG, G  
PATENT ASSIGNEE(S): (WELL) WELLCOME FOUND LTD  
COUNTRY COUNT: 11  
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 594223	A1	19940427	(199421)*	EN	29
R:	AT BE CH DE FR GB IT LI LU NL SE				
EP 594223	B1	20000301	(200016)	EN	
R:	AT BE CH DE FR GB IT LI LU NL SE				
DE 3650741	G	20000406	(200024)		

#### APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 594223	A1 Related to	EP 1986-307071	19860915
		EP 1993-120947	19860915
EP 594223	B1 Div ex	EP 1986-307071	19860915
		EP 1993-120947	19860915
DE 3650741	G	DE 1986-3650741	19860915
		EP 1993-120947	19860915

#### FILING DETAILS:

PATENT NO	KIND	PATENT NO
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PRIORITY APPLN. INFO: GB 1985-23878	19850927; US
1985-776899	19850917; GB
1986-3447	19860212; GB
1986-3719	19860214; GB
1986-8272	19860404; GB
1986-15322	19860623; US
1986-877284	19860623; US
1986-877796	19860623

The glucuronidation inhibitor and/or renal excretion inhibitor is probenecid, aspirin, acetamino-phen, lorazepam, cimetidine, ranitidine, zomepirac, clofibrate, indomethacin, ketoprofen or naproxen. The nucleoside transport inhibitor is dilazep, dipyridamole, 6((4-nitrobenzoyl) thio)- 9-(b- D-ribofuranosyl)purine, papaverine, miflazine, hexobendine or lidoflazine.

L20 ANSWER 9 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 1994-137461 [17] WPIDS  
 CROSS REFERENCE: 1986-253146 [39]; 1987-095565 [14]; 1994-168911 [21]  
 DOC. NO. CPI: C1994-063529  
 TITLE: Use of 3'-azido-3'-deoxy-thymidine and derivs. - for  
 treatment of prophylaxis of Kaposi's sarcoma, feline  
 leukaemia, multiple sclerosis or thrombocytopenia  
 purpura..  
 DERWENT CLASS: B02 B03 C02  
 INVENTOR(S): BARRY, D W; CLEMONS, R H; DE, MIRANDA P M; FREEMAN, G A;  
 FURMAN, P A; KING, D H; KIRK, L E; LEHRMAN, S N; RIDEOUT,  
 J L; SHAVER, S R; ST, CLAIR M H; WHITE, G;  
 WOLBERG, G C; ZIMMERMAN, T P; MARHT, A H; DE, MIRANDA R M  
 PATENT ASSIGNEE(S): (WELL) WELLCOME FOUND LTD  
 COUNTRY COUNT: 13  
 PATENT INFORMATION:

APPLICATION DETAILS:

Page 44

EP 594224	A2 Related to	EP 1986-307071	19860915
		EP 1993-120948	19860915
EP 594224	A3	EP 1993-120948	19860915
JP 07080898	B2 Div ex	JP 1986-59072	19860317
		JP 1988-73488	19860317
PH 26645	A Div ex	PH 1986-34252	19860915
		PH 1988-37954	19881220
PH 26859	A Div ex	PH 1986-34252	19860915
		PH 1988-37955	19881220
JP 2523527	B2	JP 1986-217871	19860916

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 594224	A3 Related to	EP 217580
JP 07080898	B2 Based on	JP 63290895
JP 2523527	B2 Previous Publ.	JP 62103100

PRIORITY APPLN. INFO: US 1986-877796 19860623; US  
 1985-776899 19850917; GB  
 1985-23878 19850927; GB  
 1986-3447 19860212; GB  
 1986-3719 19860214; GB  
 1986-8272 19860404; GB  
 1986-15322 19860623; US  
 1986-877284 19860623; GB  
 1985-6869 19850316; GB  
 1985-11774 19850509; GB  
 1985-23881 19850927; GB  
 1986-3450 19860212

AB EP 594224 A UPAB: 19970502

(1) 3-Azido-nucleosides of formula (I) and their derivs. are new. A'=purine or pyrimidine base linked at the 9- or 1-position, other than (a) (I) where A'=adenine, guanine, uridine, cytidine or thymine base, and their 5'-mono and 5'-triphosphate esters, (b) the 5'-O-acetate, 5'-O-trityl and 5'-O-(4-methylbenzenesulphonate) derivs. of cpds. where A'=uridine base and the 3'-N3 is in the erythro configuration; (c) (I) where A'=5-bromovinyluridine or 5-trifluoromethyluridine residue and the 3'-N3 is in the erythro configuration; or A'=uridine residue and the 3'-N3 is in the threo configuration; or A'=uridine residue and the 3'-N3 is in the threo configuration; or A'=5-iodo- or 5-fluoro-uridine residue and the 3'-N3 is in the erythro or threo configuration; and the 5' -O-trityl derivs. of such cpds.; (d) (I) where A'=5-bromovinyl uridine or cytidine residue and the 3'-N3 is in the threo configuration or A'=5-fluorocytidine residue and the 3'-N3 is in the erythro configuration; or A'=5-methylcytidine residue and the 3'-N3 is in the threo or erythro configuration; (e) the 5'-O-acetate of (I) where A'=4-chloro-2(1H)-pyrimidone or 4-(1H-1,2,4-triazol-1-yl)-2(1H)-pyrimidone (opt. 5-substd. by F or Me) and the 3'-N3 is in the erythro configuration; (f) the 5'-O-((4-methoxyphenyl) diphenylmethyl) derivative of (I) where A'=cytidine residue and the 3'-N3 is in the erythro configuration; and (g) the 5'-O-trityl derivative of (I) where A'=adenine residue and the 3'-N3 gp. is in the threo configuration. (2) 3-Azido nucleosides of formula (II) and their derivs. are new for use in human or veterinary therapy.

A=purine or pyrimidine base, other than thymine, linked at the 9-or 1-position.

USE/ADVANTAGE - (I) and (II) are useful in the therapy of viral and bacterial infections, especially retroviral infections,

including HIV **infections**, and **infections** caused by Gram-negative bacteria, including strains resistant to commonly used antibacterial agents.

Dwg.0/0

L20 ANSWER 10 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 1990-262694 [35] WPIDS  
 DOC. NO. CPI: C1990-113748  
 TITLE: Compsn. for treating bacterial **infections** in small domestic animals - comprises 2,4-di amino-5-(8-di methylamino-7-methyl-5-quinolyl-methyl)-pyrimidine and sulpha-di methoxine or their salts.  
 DERWENT CLASS: B03 C02  
 INVENTOR(S): **WHITE, G C; WHITE, G**  
 PATENT ASSIGNEE(S): (PITM) COOPERS ANIMAL HEALTH LTD; (WHIT-I) WHITE G; (PITM) PITMAN MOORE LTD  
 COUNTRY COUNT: 18  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 384722	A	19900829	(199035)*		
R: AT BE CH DE ES FR GB GR IT LI LU NL SE					
AU 9050002	A	19900830	(199042)		
CA 2010584	A	19900822	(199045)		
ZA 9001320	A	19911030	(199148)		
EP 384722	B1	19930908	(199336)	EN	16
R: AT BE CH DE DK ES FR GB GR IT LI LU NL SE					
DE 69003126	E	19931014	(199342)		
ES 2058784	T3	19941101	(199444)		
IE 65921	B	19951129	(199606)		

#### APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 384722	A	EP 1990-301842	19900221
ZA 9001320	A	ZA 1990-1320	19900221
EP 384722	B1	EP 1990-301842	19900221
DE 69003126	E	DE 1990-603126	19900221
		EP 1990-301842	19900221
ES 2058784	T3	EP 1990-301842	19900221
IE 65921	B	IE 1990-624	19900221

#### FILING DETAILS:

PATENT NO	KIND	PATENT NO
DE 69003126	E Based on	EP 384722
ES 2058784	T3 Based on	EP 384722

PRIORITY APPLN. INFO: GB 1989-3978 19890222

AB EP 384722 A UPAB: 19930928

Compsn. comprises 2,4-diamino-5-(8-dimethylamino-7 methyl-5-quinolylmethyl)pyrimidine and sulphadimethoxine or their salts.

USE/ADVANTAGE - Useful for treating bacterial **infections** in small domestic animals. The compsn. can be administered parenterally or orally. The cpds. are in a ratio of 1:5. In the form of tablet, the compsn. comprises 2-20 mg of the pyrimidine and 10-100 mg of sulphadimethoxine or 10-200 mg of the pyrimidine and 50-1000 mg of

sulphodimethoxine. The two components together provides a synergistic compsn. used for treating pathogenic bacteria, with the pharmacokinetic properties of the components well matched and low-dosage regime is opt.. @ 0/0

L20 ANSWER 11 OF 11 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 1987-095565 [14] WPIDS  
 CROSS REFERENCE: 1986-253146 [39]; 1994-137461 [17]; 1994-168911 [21]  
 DOC. NO. CPI: C1987-039723  
 TITLE: New and known 3-azido-nucleoside(s) - useful for therapy of viral and bacterial **infections** especially HIV retro-viral **infections** and resistant gram negative bacterial **infections**.  
 DERWENT CLASS: B02 B03 C02  
 INVENTOR(S): BARRY, D W; CLEMONS, R H; DE, MIRANDA P M; FREEMAN, G A; FURMAN, P A; KING, D H; KIRK, L E; LEHRMAN, S N; RIDEOUT, J L; SHAVER, S R; ST, CLAIR M H; **WHITE, G**; WOLBERG, G; ZIMMERMAN, T P; STCLAIR, M H; MARHT, A H; WOLBERG, G C  
 PATENT ASSIGNEE(S): (WELL) WELLCOME FOUND LTD; (WELL) BURROUGHS WELLCOME CO  
 COUNTRY COUNT: 23  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 217580	A	19870408	(198714)*	EN	96
R: AT BE CH DE FR GB IT LI LU NL SE					
GB 2181128	A	19870415	(198715)		
AU 8662702	A	19870319	(198718)		
FI 8603729	A	19870318	(198727)		
HU 42503	T	19870728	(198733)		
DK 8604417	A	19870318	(198745)		
PT 83375	A	19871020	(198746)		
DD 251984	A	19871202	(198817)		
DD 262802	A	19881214	(198920)		
ES 2002342	A	19880801	(198926)		
ES 2006672	A	19890501	(198943)		
US 5086044	A	19920204	(199208)		5
IL 80035	A	19920216	(199220)		
IL 93223	A	19920216	(199220)		
CA 1302263	C	19920602	(199228)		
DK 9101987	A	19911210	(199231)		
US 5145840	A	19920908	(199239)		12
EP 306597	A3	19920819	(199337)		
DK 167377	B	19931025	(199348)		
KR 9203804	B1	19920515	(199348)		
FI 90664	B	19931130	(199351)		
JP 2523527	B2	19960814	(199637)		36
DK 175122	B	20040607	(200438)		
DK 175192	B	20040705	(200445)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 217580	A	EP 1986-307071	19860915
GB 2181128	A	GB 1986-22194	19860915
ES 2002342	A	ES 1986-1910	19860915
ES 2006672	A	ES 1988-1870	19880616
US 5086044	A	US 1990-510590	19900418

IL 80035	A	IL 1986-80035	19860915
IL 93223	A	IL 1986-93223	19860915
CA 1302263	C	CA 1986-518308	19860916
US 5145840	A Cont of	US 1986-877284	19860623
		US 1991-679236	19910402
EP 306597	A3	EP 1988-101795	19860314
DK 167377	B	DK 1986-4417	19860915
KR 9203804	B1	KR 1986-7734	19860915
FI 90664	B	FI 1986-3729	19860915
JP 2523527	B2	JP 1986-217871	19860916
DK 175122	B	DK 1991-2026	19911218
DK 175192	B	DK 1991-2027	19911218

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
IL 93223	A Div ex	IL 80035
DK 167377	B Previous Publ.	DK 8604417
FI 90664	B Previous Publ.	FI 8603729
JP 2523527	B2 Previous Publ.	JP 62103100
DK 175122	B Previous Publ.	DK 9102026
DK 175192	B Previous Publ.	DK 9102027

PRIORITY APPLN. INFO: US 1986-877796 19860623; US

1985-776899	19850917; GB
1985-23878	19850927; GB
1986-3447	19860212; GB
1986-3719	19860214; GB
1986-8272	19860404; GB
1986-15322	19860623; US
1986-877284	19860623; GB
1986-22194	19860915; US
1990-510590	19900418; US
1991-679236	19910402; GB
1985-6869	19850316; GB
1985-11774	19850509; GB
1985-23881	19850927; GB
1986-3450	19860212

AB EP 217580 A UPAB: 20040716

(1) 3-Azido-nucleosides of formula (I) and their derivs. are new. A'=purine or pyrimidine base linked at the 9- or 1-position, other than (a) (I) where A'=adenine, guanine, uridine, cytidine or thymine base, and their 5'-mono and 5'-triphosphate esters, (b) the 5'-O-acetate, 5'-O-trityl and 5'-O-(4-methylbenzenesulphonate) derivs. of cpds. where A'=uridine base and the 3'-N3 is in the erythro configuration; (c) (I) where A'=5-bromovinyluridine or 5-trifluoromethyluridine residue and the 3'-N3 is in the erythro configuration; or A'=uridine residue and the 3'-N3 is in the threo configuration; or A'=uridine residue and the 3'-N3 is in the threo configuration; or A'=5-iodo- or 5-fluoro-uridine residue and the 3'-N3 is in the erythro or threo configuration; and the 5' -O-trityl derivs. of such cpds.; (d) (I) where A'=5-bromovinyl uridine or cytidine residue and the 3'-N3 is in the threo configuration or A'=5-fluorocytidine residue and the 3'-N3 is in the erythro configuration; or A'=5-methylcytidine residue and the 3'-N3 is in the threo or erythro configuration; (e) the 5'-O-acetate of (I) where A'=4-chloro-2(1H)-pyrimidone or 4-(1H-1,2,4-triazol-1-yl)-2(1H)-pyrimidone (opt. 5-substd. by F or Me) and the 3'-N3 is in the erythro configuration; (f) the 5'-O-((4-methoxyphenyl) diphenylmethyl) derivative of (I) where A'=cytidine residue and the 3'-N3 is in the erythro configuration; and (g) the



5'-O-trityl derivative of (I) where A'=adenine residue and the 3'-N3 gp. is in the threo configuration. (2) 3-Azido nucleosides of formula (II) and their derivs. are new for use in human or veterinary therapy.

A=purine or pyrimidine base, other than thymine, linked at the 9-or 1-position.

USE/ADVANTAGE - (I) and (II) are useful in the therapy of viral and bacterial infections, especially retroviral infections, including HIV infections, and infections caused by Gram-negative bacteria, including strains resistant to commonly used antibacterial agents.

Dwg.0/0

L21 ANSWER 1 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2004-710398 [70] WPIDS  
 DOC. NO. CPI: C2004-250569  
 TITLE: Vaccine composition useful for treating or preventing group A Streptococcal infection in individual, comprises one or more of group A Streptococcal antigen and proteosome adjuvant.  
 DERWENT CLASS: B04 D16  
 INVENTOR(S): BATZLOFF, M R; BURT, D S; GOOD, M F; LEANDERSON, T B; LOWELL, G H; WHITE, G L  
 PATENT ASSIGNEE(S): (BURT-I) BURT D; (COUN-N) COUNCIL QUEENSLAND INST MEDICAL RES; (LEAN-I) LEANDERSON T; (LOWE-I) LOWELL G; (WHIT-I) WHITE G; (IDBI-N) ID BIOMEDICAL CORP QUEBEC  
 COUNTRY COUNT: 2  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
AU 2002302132	A1	20040603	(200470)*		63
US 2005002956	A1	20050106	(200504)		

#### APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
AU 2002302132	A1	AU 2002-302132	20021115
US 2005002956	A1 Provisional	US 2002-426409P	20021115
		US 2003-706275	20031113

PRIORITY APPLN. INFO: AU 2002-302132 20021115

AB AU2002302132 A UPAB: 20041101

NOVELTY - A vaccine composition (I) comprises one or more of group A Streptococcal antigen and a proteosome adjuvant.

ACTIVITY - Antibacterial.

MECHANISM OF ACTION - Vaccine (claimed). Immunogenicity of the J14 peptide/proteosome adjuvant was determined in the murine model. The potential of intranasal immunization with the vaccine candidate and degree of protection inferred against intraperitoneal challenge were determined. Peptides complexed with proteosome adjuvant were used for intranasal immunization of Swiss outbred mice. Two boosts of 60 micro g peptide/30 micro l/mouse at 21 day intervals, followed the primary immunization. One day prior to the boosts and 15 days after serum IgG titres specific for J14 were measured and isotyping performed. IgA titres from salivations collected 2 days prior and 14 days after boosts were determined. Mice were

intraperitoneally challenged with a dose of M1 group A streptococcal (GAS) reference strain. Peptide-proteosome adjuvant group induced significant J14 specific serum antibody titres. Overall, the average titres of the control groups were not significant compared to the J14-adjuvant groups in the final bleed. Approximately, one third of group 4 (mice immunized with the carboxyl terminal anchored J14) induced antibodies that recognized the J14 peptide.

USE - (I) is useful for the treatment or prophylaxis of a group A Streptococcal **infection** in an individual which involves administering (I) to the individual intranasally, where the treatment is effected through reduction or prevention of bacterial colonization of the throat (claimed).

Dwg.0/28

L21 ANSWER 2 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2003-671302 [63] WPIDS  
 DOC. NO. NON-CPI: N2003-536060  
 DOC. NO. CPI: C2003-183016  
 TITLE: Intraluminal stent or graft useful for treating atherosclerosis comprises a tubular body including several unit cells, extends from proximal to distal end, and is capable of expanding from a radially compressed state to expanded state.  
 DERWENT CLASS: A96 B05 D22 P32  
 INVENTOR(S): WHITE, G H  
 PATENT ASSIGNEE(S): (WHIT-I) WHITE G H  
 COUNTRY COUNT: 102  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003053284	A1	20030703	(200363)*	EN	23
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU					
MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK					
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR					
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT					
RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA					
ZM ZW					
AU 2002351883	A1	20030709	(200428)		
US 2005182477	A1	20050818	(200555)		

#### APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003053284	A1	WO 2002-AU1757	20021220
AU 2002351883	A1	AU 2002-351883	20021220
US 2005182477	A1	WO 2002-AU1757	20021220
		US 2005-499016	20050425

#### FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002351883	A1 Based on	WO 2003053284

PRIORITY APPLN. INFO: AU 2001-9694 20011220; AU  
 2001-9693 20011220  
 AB WO2003053284 A UPAB: 20031001

NOVELTY - An intraluminal stent (S1) or graft (G1) comprises a tubular body extending from proximal to distal end, is capable of expanding or being expanded from a radially compressed state to a radially expanded state, and includes several unit cells. Each unit cell has a first and a second end portion, with greater dimension of the first end portion, and each optionally comprising tapering regions and a longitudinal and a traverse axis.

DETAILED DESCRIPTION - An intraluminal stent (S1) comprises a tubular body extending from proximal to distal end, and is capable of expanding or being expanded from a radially compressed state to a radially expanded state. (S1) comprises several unit cells and (G1) is circumferentially reinforced along at least a part of its length by several unit cells. Each unit cell has either a first end portion and a second end portion, where the first end portion has greater dimensions than the second end portion, and a longitudinal axis and a traverse axis, where each unit cell is symmetrical about its longitudinal axis and asymmetrical about the traverse axis; or each unit cell comprises a first end portion comprising several tapering regions and a second end portion comprising at least one tapering region.

An INDEPENDENT CLAIM is also included for a method of positioning (S1) or (G1) in a vessel of a patient involving:

- (a) introducing a catheter or other delivery device into a vein, artery or other vessel in the body of a patient when the tubular body of (S1) or (G1) is in its radially compressed state;
- (b) causing (S1) or (G1) to be carried through the catheter or other delivery device to a target site of stenosis in a vessel;
- (c) causing or allowing the tubular of the intraluminal stent to expand within the vessel; and
- (d) withdrawing the catheter or other delivery device along with any other apparatus used to introduce (S1) or (G1) into the vessel from the body of the patient.

USE - For treating stenosis or other conditions of the visceral arteries such as renal and mesenteric arteries, the iliac artery and the sub-clavian artery and stenotic lesions in the peripheral vasculature, the coronary circulation, the hepato-biliary and genito-urinary tracts; and aneurysmal disease of arteries of a patient including the aorta, renal and mesenteric arteries, the iliac artery, the sub-clavian artery and diseases of the peripheral vasculature and the coronary circulation (all claimed) such as atherosclerosis.

ADVANTAGE - (S1) And (G1) is effective in providing a stable bridge for the flow of blood through a disease section of the vessel, and has good strength and flexibility with good expansile ratio. The graft can be packaged in a compressed form into a suitable introducer catheter while at the same time provides an expanded form of suitable diameter to engage the wall of the vessel in which it is placed. The unit cells have curved or sinusoidal shape so that any length change during radial compression of the stent or the graft is compensated for by the spring-like properties of the unit cells. The radially compressed state of the tubular body enables the delivery of the stent through an introducer catheter.

DESCRIPTION OF DRAWING(S) - The figure shows a side elevational view of the stent.

Intraluminal stent 10  
Tubular body 11  
Proximal end 12  
Distal end 13  
Unit cell 14  
    First end portion 15  
First end 16  
    Second end portion 17  
Second end 18

Tapering regions 21  
 Terminating points 22  
 Tapering region of the second end 23  
 Circumferential series 25a, 25b  
 Common sides 28a, 28b

Dwg.1/15

L21 ANSWER 3 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2003-607877 [57] WPIDS  
 DOC. NO. NON-CPI: N2003-484708  
 DOC. NO. CPI: C2003-165605  
 TITLE: Engagement device for intraluminal graft or stent has  
 main body which expands from radially compressed state to  
 expanded state such that, in expanded state, at least  
 part of the body engages wall of vessel in which the  
 device is positioned.  
 DERWENT CLASS: A96 B05 D22 P32  
 INVENTOR(S): WHITE, G H  
 PATENT ASSIGNEE(S): (WHIT-I) WHITE G H  
 COUNTRY COUNT: 103  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2003053283	A1	20030703	(200357)*	EN	31
RW: AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SI SK SL SZ TR TZ UG ZM ZW W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW					
AU 2002350277	A1	20030709	(200428)		
EP 1465550	A1	20041013	(200467)	EN	
R: AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI SK TR					
US 2005171599	A1	20050804	(200552)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2003053283	A1	WO 2002-AU1725	20021219
AU 2002350277	A1	AU 2002-350277	20021219
EP 1465550	A1	EP 2002-784919	20021219
		WO 2002-AU1725	20021219
US 2005171599	A1	WO 2002-AU1725	20021219
		US 2005-499017	20050401

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2002350277	A1 Based on	WO 2003053283
EP 1465550	A1 Based on	WO 2003053283

PRIORITY APPLN. INFO: AU 2001-9692 20011220  
 AB WO2003053283 A UPAB: 20030906  
 NOVELTY - Engagement device for an intraluminal graft or stent has a main  
 body (11) which expands from a radially compressed state to a radially

expanded state such that, when in its expanded state, at least part of the main body engages a wall of a vessel in which the engagement device is positioned. The main body has a receiving section (12) to receive and engage at least a portion of the graft or stent.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for:

(1) an intraluminal assembly for placement in a vessel (14) of a patient, which comprises an engagement device, and an intraluminal graft or stent; and

(2) a method of positioning an engagement device for an intraluminal graft or stent in a diseased vessel of a patient, which includes introducing a catheter (24) or other delivery device into a vein, artery, or other vessel of a patient until a distal end of the catheter or other delivery device is positioned at or adjacent a target region of the diseased vessel; causing the engagement device for an intraluminal graft or stent to be carried through the catheter or other delivery device to the target region, with the main body of the device in its radially compressed state; releasing the main body from the distal end of the catheter or other delivery device and causing or allowing the main body to move to its radially expanded state within the target region of the diseased vessel such that at least part of the main body engages a wall of the target region of the vessel; positioning a radially compressed intraluminal graft or stent at least partially within the receiving region of the main body; causing or allowing the intraluminal graft or stent to move to a radially expanded state such that at least a portion of the intraluminal stent or graft engages with the receiving region of the main body; and withdrawing the catheter or other delivery device along with any other apparatus used to introduce the engagement device for an intraluminal graft or stent and/or the intraluminal graft or stent from the patient.

USE - The engagement device is used for securing or sealing an intraluminal graft or stent in place within a vessel of a patient. The intraluminal graft or stent is used in the grafting or stenting of diseased arteries of a patient including the aorta and the visceral arteries e.g., the renal and mesenteric arteries, the iliac artery, and the sub-clavian artery; or of the peripheral vasculature, the coronary circulation, the hepato-biliary, and genito-urinary tracts (claimed).

ADVANTAGE - The inventive device eliminates the problems associated with conventional intraluminal grafts which do not align or seal well with a diseased or anatomically unique vessel structure and which have the tendency to slip within the vessel, causing the graft to occlude an opening of a pre- or post-branching vessel.

DESCRIPTION OF DRAWING(S) - The figure depicts delivery of the inventive device to a target vessel.

Main body 11

Receiving section 12

Vessel 14

Outer wall 16

Internal wall 17

Catheter 24

Dwg.2b/9

L21 ANSWER 4 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 2002-227039 [28] WPIDS

DOC. NO. CPI: C2002-069098

TITLE: Treating e.g. neuropathic pain, comprises administration of new and known compounds which are high potency capsaicin receptor antagonists, and are not capsaicin analogs.

DERWENT CLASS: B02 B03 B07

INVENTOR(S): BAKTHAVATCHALAM, R; DESIMONE, R W; HODGETTS, K J;  
 HUTCHINSON, A; KRAUSE, J E; **WHITE, G G**;  
 HUTCHISON, A  
 PATENT ASSIGNEE(S): (DESI-I) DESIMONE R W; (HODG-I) HODGETTS K J; (HUTC-I)  
 HUTCHISON A; (KRAU-I) KRAUSE J E; (NEUR-N) NEUROGEN CORP;  
 (WHIT-I) WHITE G G; (BAKT-I) BAKTHAVATCHALAM R  
 COUNTRY COUNT: 97  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2002008221	A2	20020131	(200228)*	EN	209
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001080667	A	20020205	(200236)		
US 2002132853	A1	20020919	(200264)		
EP 1301484	A2	20030416	(200328)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
KR 2003024799	A	20030326	(200346)		
BR 2001012631	A	20030923	(200373)		
CN 1443170	A	20030917	(200382)		
US 6723730	B2	20040420	(200427)		
JP 2004525071	W	20040819	(200455)	337	
US 2004176443	A1	20040909	(200459)		
NZ 523526	A	20041029	(200474)		
MX 2003000458	A1	20040601	(200504)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2002008221	A2	WO 2001-US22930	20010720
AU 2001080667	A	AU 2001-80667	20010720
US 2002132853	A1	US 2000-219529P	20000720
	Provisional	US 2000-230726P	20000907
	Provisional	US 2001-280223P	20010330
		US 2001-910442	20010720
EP 1301484	A2	EP 2001-959075	20010720
		WO 2001-US22930	20010720
KR 2003024799	A	KR 2003-700866	20030120
BR 2001012631	A	BR 2001-12631	20010720
		WO 2001-US22930	20010720
CN 1443170	A	CN 2001-813046	20010720
US 6723730	B2	US 2000-219529P	20000720
	Provisional	US 2000-230726P	20000907
	Provisional	US 2001-280223P	20010330
		US 2001-910442	20010720
JP 2004525071	W	WO 2001-US22930	20010720
		JP 2002-514127	20010720
US 2004176443	A1	US 2000-219529P	20000720
	Provisional	US 2000-230726P	20000907
	Provisional	US 2001-280223P	20010330
	Cont of	US 2001-910442	20010720
		US 2004-799286	20040312
NZ 523526	A	NZ 2001-523526	20010720

MX 2003000458	A1	WO 2001-US22930	20010720
		WO 2001-US22930	20010720
		MX 2003-458	20030116

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001080667	A Based on	WO 2002008221
EP 1301484	A2 Based on	WO 2002008221
BR 2001012631	A Based on	WO 2002008221
JP 2004525071	W Based on	WO 2002008221
US 2004176443	A1 Cont of	US 6723730
NZ 523526	A Based on	WO 2002008221
MX 2003000458	A1 Based on	WO 2002008221

PRIORITY APPLN. INFO: US 2001-280223P 20010330; US  
 2000-219529P 20000720; US  
 2000-230726P 20000907; US  
 2001-910442 20010720; US  
 2004-799286 20040312

AB WO 200208221 A UPAB: 20021031

NOVELTY - Treating at least one symptom selected from e.g. burns or irritation due to exposure to heat or light, bronchoconstriction or irritation due to exposure to tear gas, and neuropathic pain and peripheral nerve mediated pain comprises administering a compound that is a high potency capsaicin receptor antagonist in an in vitro assay of capsaicin receptor antagonism, and is not a capsaicin analog.

DETAILED DESCRIPTION - Treating at least one symptom selected from symptoms of exposure to capsaicin, burns or irritation due to exposure to heat or light, burns, bronchoconstriction or irritation due to exposure to tear gas, burns or irritation due to exposure to acid and treating neuropathic pain and peripheral nerve mediated pain comprises administering a dose of a compound that is a high potency capsaicin receptor antagonist in an in vitro assay of capsaicin receptor antagonism, and is not a capsaicin analog.

INDEPENDENT CLAIMS are included for compounds of formula (I)-(IV) and their salts.

A = e.g. absent or is O, S, NRA, CRBRB', NRACRBRB' or CRBRB'NRA;

RA, RB, RB' = H or alkyl;

Z = O or S;

R1, R2 = H or alkyl;

R3, R4 = e.g. alkyl, alkenyl, alkynyl, alkoxy, mono or dialkylamino, alkylthio or alkyl ketone (all optionally substituted);

Ar1, Ar2 = e.g. cycloalkyl, 5-8 membered heterocyclylalkyl (containing 1-3 heteroatoms of N, O or S) or aryl having 1-3 rings (all optionally substituted);

Ar1 asterisk, Ar2 asterisk = e.g. cyclohexyl, cyclopentyl, piperidinyl, piperazinyl, phenyl or benzo(b)thiophenyl, (all optionally substituted);

x = 1 or 3;

Ar1'', Ar2'' = e.g. cyclohexyl, cyclopentyl, piperidinyl, piperazinyl, pyrrolyl or quinolinyl, (where Ar1'' and Ar2'' are both optionally substituted);

G, Q, T, W = e.g. N or CH;

R3 asterisk, R4 asterisk = e.g. 1-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, 1-6C alkoxy, mono or di(1-6C)alkylamino or aryl having 1-3 rings, (all optionally substituted);

R5 asterisk = e.g. 3-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, 3-6C

alkoxy or NH(1-6C alkyl) (all optionally substituted); and

R9 asterisk = e.g. 1-6C alkyl, 2-6C alkenyl, 2-6C alkynyl, 1-6C alkoxy or NH(1-6C alkyl) (all optionally substituted) or halo.

The full definitions are given in the DEFINITIONS (Full Definitions) Field.

ACTIVITY - Analgesic; Vulnerary; Uropathic; Antipsoriatic; Dermatological; Antiallergic.

In an assay to assess the effectiveness of the compounds in relieving one of the symptoms of neuropathic pain produced by unilateral mononeuropathy (namely mechanical hyperalgesia), the compounds produced a reduction in mechanical hyperalgesia elicited by a pin-prick stimulus in rats with a chronic constriction injury, at doses of 50 mg/kg or less.

MECHANISM OF ACTION - Capsaicin receptor modulator; Capsaicin receptor antagonist; Vanilloid binding inhibitor.

USE - For treating at least one symptom selected from symptoms of exposure to capsaicin, burns or irritation due to exposure to heat or light, burns, bronchoconstriction or irritation due to exposure to tear gas, burns or irritation due to exposure to acid and treating neuropathic pain and peripheral nerve mediated pain (e.g. pain associated with post-mastectomy pain syndrome, stump pain, phantom limb pain, oral neuropathic pain, Charcot's pain, toothache, venomous snake bite, spider bite, insect sting, postherpetic neuralgia, diabetic neuropathy, reflex sympathetic dystrophy, trigeminal neuralgia, osteoarthritis, rheumatoid arthritis, fibromyalgia, Guillain-Barre syndrome, meralgia paresthetica, burning mouth syndrome, bilateral peripheral neuropathy, causalgia, sciatic neuritis, peripheral neuritis, polyneuritis, optic neuritis, postfebrile neuritis, migrating neuritis, segmental neuritis, Gombault's neuritis, neuronitis, cervicobrachial neuralgia, cranial neuralgia, geniculate neuralgia, glossopharyngeal neuralgia, migranous neuralgia, idiopathic neuralgia, intercostal neuralgia, mammary neuralgia, mandibular joint neuralgia, Morton's neuralgia, nasociliary neuralgia, occipital neuralgia, red neuralgia, Sluder's neuralgia, splenopalatine neuralgia, supraorbital neuralgia, vidian neuralgia, sinus headache, tension headache, labor, childbirth, intestinal gas, menstruation, cancer and trauma); and for reducing the calcium conductance of a capsaicin receptor, (all claimed). Also useful for as tools for the analysis of type I vanilloid receptors (VRI) and as probes for the quantitative measurement and localization of VRI receptors in cell and tissue samples. Also useful for reducing the frequency of **urinary** incontinence (e.g. caused by detrusor hyperflexia of spinal origin or bladder hypersensitivity) and for treating itching conditions (e.g. psoriatic pruritus, itch due to hemodialysis, aquagenic pruritus, itching associated with vulvar vestibulitis, contact dermatitis or skin allergies).

Dwg.0/0

L21 ANSWER 5 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2001-557626 [62] WPIDS  
 DOC. NO. CPI: C2001-165808  
 TITLE: New improved vaccines, which comprise proteosomes and protein antigens, useful against influenza, particularly for protecting humans against influenza **infection**  
 .  
 DERWENT CLASS: B04 D16  
 INVENTOR(S): BURT, D S; FRIES, L F; JONES, D H; LOWELL, G H; PLANTE, M; TOROSSIAN, K; **WHITE, G L**  
 PATENT ASSIGNEE(S): (INTE-N) INTELLIVAX INT INC; (IDBI-N) ID BIOMEDICAL CORP QUEBEC; (BURT-I) BURT D S; (FRIE-I) FRIES L F; (JONE-I) JONES D H; (LOWE-I) LOWELL G H; (PLAN-I) PLANTE M; (TORO-I) TOROSSIAN K; (WHIT-I) WHITE G L  
 COUNTRY COUNT: 95



## PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001060402	A2	20010823	(200162)*	EN	44
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
AU 2001038478	A	20010827	(200176)		
US 2001053368	A1	20011220	(200206)		
NO 2002003829	A	20020813	(200277)		
EP 1255561	A2	20021113	(200282)	EN	
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI TR					
JP 2003522802	W	20030729	(200358)		66
EP 1419784	A2	20040519	(200433)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE TR					
US 6743900	B2	20040601	(200436)		
US 2004156867	A1	20040812	(200454)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001060402	A2	WO 2001-US5222	20010215
AU 2001038478	A	AU 2001-38478	20010215
US 2001053368	A1 Provisional	US 2000-182476P	20000215
		US 2001-788280	20010215
NO 2002003829	A	WO 2001-US5222	20010215
		NO 2002-3829	20020813
EP 1255561	A2	EP 2001-910923	20010215
		WO 2001-US5222	20010215
JP 2003522802	W	JP 2001-559498	20010215
		WO 2001-US5222	20010215
EP 1419784	A2 Div ex	EP 2001-910923	20010215
		EP 2004-953	20010215
US 6743900	B2 Provisional	US 2000-182476P	20000215
		US 2001-788280	20010215
US 2004156867	A1 Provisional	US 2000-182476P	20000215
	Div ex	US 2001-788280	20010215
		US 2004-771737	20040203

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001038478	A Based on	WO 2001060402
EP 1255561	A2 Based on	WO 2001060402
JP 2003522802	W Based on	WO 2001060402
EP 1419784	A2 Div ex	EP 1255561
US 2004156867	A1 Div ex	US 6743900

PRIORITY APPLN. INFO: US 2000-182476P 20000215; US  
2001-788280 20010215; US  
2004-771737 20040203

AB WO 200160402 A UPAB: 20011026  
NOVELTY - An influenza vaccine, which comprises at least one influenza

hemagglutinin (HA) formulated with proteosomes in the substantial absence of detergent, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) preparing a vaccine effective against (viral) **infection**, comprising:

(a) providing a mixture of at least one (viral) protein antigen with a proteosome preparation in the presence of detergent;

(b) removing detergent from the mixture by diafiltration or ultrafiltration to obtain a proteosome-antigen composition; and

(c) formulating the composition into a vaccine;

(2) a vaccine prepared by the method of (1);

(3) preparing a multivalent vaccine effective against viral **infection**, comprising:

(a) providing a mixture of at least two viral protein antigens to a proteosome preparation in the presence of detergent, and employing steps (b) and (c) of (1); or

(b) mixing compositions, each containing at least one protein antigen prepared in (1) and formulating the mixture into a vaccine;

(4) eliciting an immune response against influenza in a subject comprising administering to the subject the vaccine; and

(5) improved method for preparing proteosomes, where the improvement, comprises:

(a) performing one or more steps comprising precipitation in the presence of ethanol followed by extraction with 0.1 % detergent solution; or

(b) omitting precipitation by ammonium sulfate.

ACTIVITY - Virucide.

No biological data is given.

MECHANISM OF ACTION - Vaccine.

USE - The vaccine is useful against influenza, particularly for protecting against influenza **infection**, especially in human

(claimed) The methods are useful for preparing vaccine preparations.

Dwg.0/7

L21 ANSWER 6 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 2001-328586 [34] WPIDS  
 CROSS REFERENCE: 2001-061819 [07]  
 DOC. NO. NON-CPI: N2001-236469  
 TITLE: Intraluminal graft for use in treating aneurysmal or stenotic disease, has tubular body of predetermined, non linear shape.  
 DERWENT CLASS: P32  
 INVENTOR(S): DEHDASHTIAN, M; JIMINEZ, T; WHITE, G H; YU, W; JIMENEZ, T S; WHITE, G  
 PATENT ASSIGNEE(S): (EDWA-N) EDWARDS LIFESCIENCES CORP; (ENDO-N) ENDOGAD RES PTY LTD; (EDWA-N) EDWARDS LIFESCIENCES LLC; (WHIT-I) WHITE G; (YUWW-I) YU W  
 COUNTRY COUNT: 91  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001030270	A2	20010503	(200134)*	EN	21
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ					
NL OA PT SD SE SL SZ TZ UG ZW					
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM					
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC					
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE					
SG SI SK SL TJ TM TR TT TZ UA UG					

AU 2001037889 A 20010508 (200149)  
 EP 1214021 A2 20020619 (200240) EN  
 R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
 RO SE SI  
 US 2003033002 A1 20030213 (200314)  
 AU 778349 B2 20041202 (200506)  
 US 6849088 B2 20050201 (200511)#  
 US 2006015176 A1 20060119 (200607)

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001030270	A2	WO 2000-US26239	20000925
AU 2001037889	A	AU 2001-37889	20000925
EP 1214021	A2	EP 2000-991996	20000925
		WO 2000-US26239	20000925
US 2003033002	A1 CIP of	US 1998-163580	19980930
	CIP of	US 1998-204699	19981203
		US 2001-55787	20011107
AU 778349	B2	AU 2001-37889	20000925
US 6849088	B2 CIP of	US 1998-163580	19980930
	CIP of	US 1998-204699	19981203
		US 2001-55787	20011107
US 2006015176	A1 Cont of	US 2000-595043	20000615
		US 2005-173322	20050630

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
AU 2001037889	A Based on	WO 2001030270
EP 1214021	A2 Based on	WO 2001030270
US 2003033002	A1 CIP of	US 6368345
AU 778349	B2 Previous Publ.	AU 2001037889
	Based on	WO 2001030270
US 6849088	B2 CIP of	US 6368345

PRIORITY APPLN. INFO: AU 1999-3029 19990923

AB WO 200130270 A UPAB: 20060130

NOVELTY - An intraluminal graft (10) comprises of a tubular body (22) of a predetermined, non linear shape. The graft can be introduced into e.g. an aortic aneurysm (11) in the aorta (12) from a catheter through one of the femoral arteries (16) in the aorta. The tubular body shape allows the graft to conform to the natural or pathological contours of the aorta.

DETAILED DESCRIPTION - The tubular body is preferably formed from woven Dacron, interwoven with spaced apart reinforcing wires. An INDEPENDENT CLAIM is also included for a method for emplacing an intraluminal device.

USE - For treating aneurysms of e.g. femoral artery, popliteal artery, thoracic segment of aorta, or visceral arteries e.g. renal and mesenteric arteries, iliac artery, sub clavian artery. Also for treating stenotic lesions in peripheral vasculature.

ADVANTAGE - Graft can be used for treating stenotic lesions in other vessels e.g. hepato biliary and genito urinary tracts, or vessels which constitute coronary circulation. Graft can conform or securely fit into wall of vessel e.g. aorta, without possible dislodging, when aneurysm in aorta expands. Graft can also align with both non linear and substantially linear vessels. Allows vessel with graft to be imaged by e.g. ultrasound, plain abdominal film, computerized tomography CT

scanning. Enhances patient's vasculature.

DESCRIPTION OF DRAWING(S) - The figure shows the partially cut away ventral view of the patient, with the intraluminal graft bridging the aortic aneurysm of the patient. Another figure shows the alternate embodiment of the intraluminal graft.

Intraluminal graft 10

Aortic aneurysm 11

Aorta 12

Femoral arteries 16

Tubular body 22

Dwg.1,9/9

L21 ANSWER 7 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 1994-168911 [21] WPIDS  
 CROSS REFERENCE: 1986-253146 [39]; 1987-095565 [14]; 1994-137461 [17]  
 DOC. NO. CPI: C1994-077302  
 TITLE: New therapeutic combination of 3'-azido-3'-deoxy  
 thymidine and a second agent - useful in the treatment of  
 human retroviral **infection** especially HTLV-1, HTLV-2  
 and HIV **infections**..  
 DERWENT CLASS: B03 B05  
 INVENTOR(S): BARRY, D W; CLEMONS, R H; DE MIRANDA, P M; FREEMAN, G A;  
 FURMAN, P A; KING, D H; KIRK, L E; LEHRMAN, S N; RIDEOUT,  
 J L; SHAVER, S R; ST CLAIR, M H; **WHITE, G**;  
 WOLBERG, G C; ZIMMERMAN, T P; WOLBERG, G  
 PATENT ASSIGNEE(S): (WELL) WELLCOME FOUND LTD  
 COUNTRY COUNT: 11  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 594223	A1	19940427	(199421)*	EN	29
R:	AT BE CH DE FR GB IT LI LU NL SE				
EP 594223	B1	20000301	(200016)	EN	
R:	AT BE CH DE FR GB IT LI LU NL SE				
DE 3650741	G	20000406	(200024)		

#### APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 594223	A1 Related to	EP 1986-307071	19860915
		EP 1993-120947	19860915
EP 594223	B1 Div ex	EP 1986-307071	19860915
		EP 1993-120947	19860915
DE 3650741	G	DE 1986-3650741	19860915
		EP 1993-120947	19860915

#### FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 594223	B1 Div ex	EP 217580
DE 3650741	G Based on	EP 594223

PRIORITY APPLN. INFO: GB 1985-23878 19850927; US  
 1985-776899 19850917; GB  
 1986-3447 19860212; GB  
 1986-3719 19860214; GB  
 1986-8272 19860404; GB

1986-15322 19860623; US  
 1986-877284 19860623; US  
 1986-877796 19860623

AB EP 594223 A UPAB: 20000522  
 Therapeutic combination (A) comprises 3'-azido -3'-deoxy -thymidine (AZT) and at least one of glucuronidation inhibitor and or renal excretion inhibitor, nucleoside transport inhibitor, therapeutic nucleoside, antibacterial agent, interferon, interleukin II, suramin, phosphonoformate, HPA 23 and a 2',3'-dideoxy nucleoside.

The glucuronidation inhibitor and/or renal excretion inhibitor is probenecid, aspirin, acetamino-phen, lorazepam, cimetidine, ranitidine, zomepirac, clofibrate, indomethacin, ketoprofen or naproxen. The nucleoside transport inhibitor is dilazep, dipyridamole, 6((4-nitrobenzoyl) thio)- 9-(b- D-ribofuranosyl)purine, papaverine, mioflazine, hexobendine or lidoflazine.

USE - (A) is useful in the treatment or prophylaxis of a human retroviral **infection** especially HTLV or HIV **infection**. More especially HTLV-1 or HTLV-11 **infection**, and is therefore useful in the treatment of prophylaxis of AIDS, AIDS-related complex, persistent generalised lymphadenopathy and asymptomatic AIDS-carrier state.  
 Dwg.0/0

L21 ANSWER 8 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 1994-137461 [17] WPIDS  
 CROSS REFERENCE: 1986-253146 [39]; 1987-095565 [14]; 1994-168911 [21]  
 DOC. NO. CPI: C1994-063529  
 TITLE: Use of 3'-azido-3'-deoxy-thymidine and derivs. - for treatment of prophylaxis of Kaposi's sarcoma, feline leukaemia, multiple sclerosis or thrombocytopenia purpura..  
 DERWENT CLASS: B02 B03 C02  
 INVENTOR(S): BARRY, D W; CLEMONS, R H; DE, MIRANDA P M; FREEMAN, G A; FURMAN, P A; KING, D H; KIRK, L E; LEHRMAN, S N; RIDEOUT, J L; SHAVER, S R; ST, CLAIR M H; **WHITE, G**; WOLBERG, G C; ZIMMERMAN, T P; MARHT, A H; DE, MIRANDA R M  
 PATENT ASSIGNEE(S): (WELL) WELLCOME FOUND LTD  
 COUNTRY COUNT: 13  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 594224	A2	19940427	(199417)*	EN	9
	R:	AT BE CH DE FR GB IT LI LU NL SE			
EP 594224	A3	19940713	(199528)		
JP 07080898	B2	19950830	(199539)		13
PH 26645	A	19920819	(199634)		
PH 26859	A	19921116	(199634)		
JP 2523527	B2	19960814	(199637)		36

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 594224	A2 Related to	EP 1986-307071	19860915
		EP 1993-120948	19860915
EP 594224	A3	EP 1993-120948	19860915
JP 07080898	B2 Div ex	JP 1986-59072	19860317
		JP 1988-73488	19860317
PH 26645	A Div ex	PH 1986-34252	19860915
		PH 1988-37954	19881220

PH 26859	A Div ex	PH 1986-34252	19860915
		PH 1988-37955	19881220
JP 2523527	B2	JP 1986-217871	19860916

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
EP 594224	A3 Related to	EP 217580
JP 07080898	B2 Based on	JP 63290895
JP 2523527	B2 Previous Publ.	JP 62103100

PRIORITY APPLN. INFO: US 1986-877796 19860623; US  
 1985-776899 19850917; GB  
 1985-23878 19850927; GB  
 1986-3447 19860212; GB  
 1986-3719 19860214; GB  
 1986-8272 19860404; GB  
 1986-15322 19860623; US  
 1986-877284 19860623; GB  
 1985-6869 19850316; GB  
 1985-11774 19850509; GB  
 1985-23881 19850927; GB  
 1986-3450 19860212

AB EP 594224 A UPAB: 19970502

(1) 3-Azido-nucleosides of formula (I) and their derivs. are new. A'=purine or pyrimidine base linked at the 9- or 1-position, other than (a) (I) where A'=adenine, guanine, uridine, cytidine or thymine base, and their 5'-mono and 5'-triphosphate esters, (b) the 5'-O-acetate, 5'-O-trityl and 5'-O-(4-methylbenzenesulphonate) derivs. of cpds. where A'=uridine base and the 3'-N3 is in the erythro configuration; (c) (I) where A'=5-bromovinyluridine or 5-trifluoromethyluridine residue and the 3'-N3 is in the erythro configuration; or A'=uridine residue and the 3'-N3 is in the threo configuration; or A'=5-iodo- or 5-fluoro-uridine residue and the 3'-N3 is in the erythro or threo configuration; and the 5' -O-trityl derivs. of such cpds.; (d) (I) where A'=5-bromovinyl uridine or cytidine residue and the 3'-N3 is in the threo configuration or A'=5-fluorocytidine residue and the 3'-N3 is in the erythro configuration; or A'=5-methylcytidine residue and the 3'-N3 is in the threo or erythro configuration; (e) the 5'-O-acetate of (I) where A'=4-chloro-2(1H)-pyrimidone or 4-(1H-1,2,4-triazol-1-yl)-2(1H)-pyrimidone (opt. 5-substd. by F or Me) and the 3'-N3 is in the erythro configuration; (f) the 5'-O-((4-methoxyphenyl) diphenylmethyl) derivative of (I) where A'=cytidine residue and the 3'-N3 is in the erythro configuration; and (g) the 5'-O-trityl derivative of (I) where A'=adenine residue and the 3'-N3 gp. is in the threo configuration. (2) 3-Azido nucleosides of formula (II) and their derivs. are new for use in human or veterinary therapy.

A=purine or pyrimidine base, other than thymine, linked at the 9-or 1-position.

USE/ADVANTAGE - (I) and (II) are useful in the therapy of viral and bacterial **infections**, especially retroviral **infections**, including HIV **infections**, and **infections** caused by Gram-negative bacteria, including strains resistant to commonly used antibacterial agents.

Dwg.0/0

L21 ANSWER 9 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN  
 ACCESSION NUMBER: 1990-262694 [35] WPIDS  
 DOC. NO. CPI: C1990-113748

TITLE: Compsn. for treating bacterial **infections** in small domestic animals - comprises 2,4-di amino-5-(8-di methylamino-7-methyl-5-quinolyl-methyl)-pyrimidine and sulpha-di methoxine or their salts.

DERWENT CLASS: B03 C02

INVENTOR(S): **WHITE, G C; WHITE, G**

PATENT ASSIGNEE(S): (PITM) COOPERS ANIMAL HEALTH LTD; (WHIT-I) WHITE G; (PITM) PITMAN MOORE LTD

COUNTRY COUNT: 18

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 384722	A	19900829	(199035)*		
	R:	AT BE CH DE ES FR GB GR IT LI LU NL SE			
AU 9050002	A	19900830	(199042)		
CA 2010584	A	19900822	(199045)		
ZA 9001320	A	19911030	(199148)		
EP 384722	B1	19930908	(199336)	EN	16
	R:	AT BE CH DE DK ES FR GB GR IT LI LU NL SE			
DE 69003126	E	19931014	(199342)		
ES 2058784	T3	19941101	(199444)		
IE 65921	B	19951129	(199606)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 384722	A	EP 1990-301842	19900221
ZA 9001320	A	ZA 1990-1320	19900221
EP 384722	B1	EP 1990-301842	19900221
DE 69003126	E	DE 1990-603126	19900221
		EP 1990-301842	19900221
ES 2058784	T3	EP 1990-301842	19900221
IE 65921	B	IE 1990-624	19900221

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
DE 69003126	E Based on	EP 384722
ES 2058784	T3 Based on	EP 384722

PRIORITY APPLN. INFO: GB 1989-3978 19890222

AB EP 384722 A UPAB: 19930928

Compsn. comprises 2,4-diamino-5-(8-dimethylamino-7 methyl-5-quinolylmethyl)pyrimidine and sulphadimethoxine or their salts.

USE/ADVANTAGE - Useful for treating bacterial **infections** in small domestic animals. The compsn. can be administered parenterally or orally. The cpds. are in a ratio of 1:5. In the form of tablet, the compsn. comprises 2-20 mg of the pyrimidine and 10-100 mg of sulphadimethoxine or 10-200 mg of the pyrimidine and 50-1000 mg of sulphodimethoxine. The two components together provides a synergistic compsn. used for treating pathogenic bacteria, with the pharmacokinetic properties of the components well matched and low-dosage regime is opt.. @ 0/0

L21 ANSWER 10 OF 10 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN

ACCESSION NUMBER: 1987-095565 [14] WPIDS

CROSS REFERENCE: 1986-253146 [39]; 1994-137461 [17]; 1994-168911 [21]

DOC. NO. CPI: C1987-039723  
 TITLE: New and known 3-azido-nucleoside(s) - useful for therapy of viral and bacterial **infections** especially HIV retro-viral **infections** and resistant gram negative bacterial **infections**.  
 DERWENT CLASS: B02 B03 C02  
 INVENTOR(S): BARRY, D W; CLEMONS, R H; DE, MIRANDA P M; FREEMAN, G A; FURMAN, P A; KING, D H; KIRK, L E; LEHRMAN, S N; RIDEOUT, J L; SHAVER, S R; ST, CLAIR M H; **WHITE, G**; WOLBERG, G; ZIMMERMAN, T P; STCLAIR, M H; MARHT, A H; WOLBERG, G C  
 PATENT ASSIGNEE(S): (WELL) WELLCOME FOUND LTD; (WELL) BURROUGHS WELLCOME CO  
 COUNTRY COUNT: 23  
 PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
EP 217580	A	19870408	(198714) *	EN	96
R: AT BE CH DE FR GB IT LI LU NL SE					
GB 2181128	A	19870415	(198715)		
AU 8662702	A	19870319	(198718)		
FI 8603729	A	19870318	(198727)		
HU 42503	T	19870728	(198733)		
DK 8604417	A	19870318	(198745)		
PT 83375	A	19871020	(198746)		
DD 251984	A	19871202	(198817)		
DD 262802	A	19881214	(198920)		
ES 2002342	A	19880801	(198926)		
ES 2006672	A	19890501	(198943)		
US 5086044	A	19920204	(199208)	5	
IL 80035	A	19920216	(199220)		
IL 93223	A	19920216	(199220)		
CA 1302263	C	19920602	(199228)		
DK 9101987	A	19911210	(199231)		
US 5145840	A	19920908	(199239)	12	
EP 306597	A3	19920819	(199337)		
DK 167377	B	19931025	(199348)		
KR 9203804	B1	19920515	(199348)		
FI 90664	B	19931130	(199351)		
JP 2523527	B2	19960814	(199637)	36	
DK 175122	B	20040607	(200438)		
DK 175192	B	20040705	(200445)		

## APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
EP 217580	A	EP 1986-307071	19860915
GB 2181128	A	GB 1986-22194	19860915
ES 2002342	A	ES 1986-1910	19860915
ES 2006672	A	ES 1988-1870	19880616
US 5086044	A	US 1990-510590	19900418
IL 80035	A	IL 1986-80035	19860915
IL 93223	A	IL 1986-93223	19860915
CA 1302263	C	CA 1986-518308	19860916
US 5145840	A	US 1986-877284	19860623
		US 1991-679236	19910402
EP 306597	A3	EP 1988-101795	19860314
DK 167377	B	DK 1986-4417	19860915
KR 9203804	B1	KR 1986-7734	19860915



FI 90664	B	FI 1986-3729	19860915
JP 2523527	B2	JP 1986-217871	19860916
DK 175122	B	DK 1991-2026	19911218
DK 175192	B	DK 1991-2027	19911218

## FILING DETAILS:

PATENT NO	KIND	PATENT NO
IL 93223	A Div ex	IL 80035
DK 167377	B Previous Publ.	DK 8604417
FI 90664	B Previous Publ.	FI 8603729
JP 2523527	B2 Previous Publ.	JP 62103100
DK 175122	B Previous Publ.	DK 9102026
DK 175192	B Previous Publ.	DK 9102027

PRIORITY APPLN. INFO: US 1986-877796 19860623; US  
 1985-776899 19850917; GB  
 1985-23878 19850927; GB  
 1986-3447 19860212; GB  
 1986-3719 19860214; GB  
 1986-8272 19860404; GB  
 1986-15322 19860623; US  
 1986-877284 19860623; GB  
 1986-22194 19860915; US  
 1990-510590 19900418; US  
 1991-679236 19910402; GB  
 1985-6869 19850316; GB  
 1985-11774 19850509; GB  
 1985-23881 19850927; GB  
 1986-3450 19860212

AB EP 217580 A UPAB: 20040716

(1) 3-Azido-nucleosides of formula (I) and their derivs. are new.  
 A'=purine or pyrimidine base linked at the 9- or 1-position, other than  
 (a) (I) where A'=adenine, guanine, uridine, cytidine or thymine base, and  
 their 5'-mono and 5'-triphosphate esters, (b) the 5'-O-acetate,  
 5'-O-trityl and 5'-O-(4-methylbenzenesulphonate) derivs. of cpds. where  
 A'=uridine base and the 3'-N3 is in the erythro configuration; (c) (I)  
 where A'=5-bromovinyluridine or 5-trifluoromethyluridine residue and the  
 3'-N3 is in the erythro configuration; or A'=uridine residue and the 3'-N3  
 is in the threo configuration; or A'=uridine residue and the 3'-N3 is in  
 the threo configuration; or A'=5-iodo- or 5-fluoro-uridine residue and the  
 3'-N3 is in the erythro or threo configuration; and the 5' -O-trityl  
 derivs. of such cpds.; (d) (I) where A'=5-bromovinyl uridine or cytidine  
 residue and the 3'-N3 is in the threo configuration or A'=5-fluorocytidine  
 residue and the 3'-N3 is in the erythro configuration; or  
 A'=5-methylcytidine residue and the 3'-N3 is in the threo or erythro  
 configuration; (e) the 5'-O-acetate of (I) where A'=4-chloro-2(1H)-  
 pyrimidone or 4-(1H-1,2,4-triazol-1-yl)-2(1H)-pyrimidone (opt. 5-substd.  
 by F or Me) and the 3'-N3 is in the erythro configuration; (f) the  
 5'-O-((4-methoxyphenyl) diphenylmethyl) derivative of (I) where A'=cytidine  
 residue and the 3'-N3 is in the erythro configuration; and (g) the  
 5'-O-trityl derivative of (I) where A'=adenine residue and the 3'-N3 gp. is in  
 the threo configuration. (2) 3-Azido nucleosides of formula (II) and their  
 derivs. are new for use in human or veterinary therapy.

A=purine or pyrimidine base, other than thymine, linked at the 9-or  
 1-position.

USE/ADVANTAGE - (I) and (II) are useful in the therapy of viral and  
 bacterial **infections**, especially retroviral **infections**,  
 including HIV **infections**, and **infections** caused by

Krishnan Ganapathy 10/691,423

Gram-negative bacteria, including strains resistant to commonly used  
antibacterial agents.  
Dwg.0/0

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